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THE
LARYNGOSCOPE.

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OSTEOMA OF THE PARANASAL SINUSES WITH REPORT OF THREE CASES.*

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New York, N. Y.

Osteoma of the nasal cavity and paranasal sinuses is a well recognized clinical entity as witnessed by the extensive literature available on the subject; however, such literature as is available is concerned primarily with the gross and clinical aspects of the lesion and its surgical treatment. It is the author's plan, therefore, to emphasize the histopathology of this fairly uncommon tumor in his review of the literature and presentation of three cases.

INCIDENCE.

Although all bones of the body may be attacked, it is generally agreed that the bones of the paranasal sinuses are the most commonly involved. The statistics of Boenninghaus, completed by Dahman[1] (1922), enumerated 234 cases. Twenty years later, Malan[2] listed 458 cases of osteoma of the paranasal sinuses. Narrowed down still further, the frontal sinus is the most frequent site for the development of osteoma of the paranasal sinuses. Of the 458 cases reported by Malan, the frontal sinus was involved in 39 per cent, the ethmoid sinus in 24 per

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cent, the maxillary sinus in 9 per cent and the sphenoid sinus in 1 to 2 per cent. The statistics of Boenninghaus produce similar percentages. Teed[3] (1941) reported that a survey of the literature revealed 321 cases of osteoma of the frontal sinus alone. Unfortunately, there has been no comprehensive survey of the cases involving the ethmoid sinuses. From a review of the literature in 1938, Rawlins[4] reported only 27

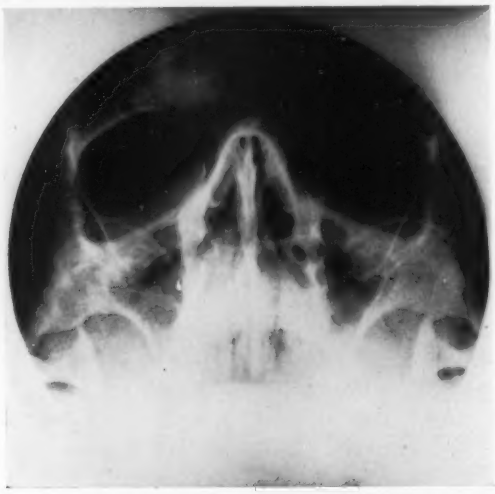

Fig. 1. Case 1. Photograph of X-ray of sinuses showing the osteoma located in the outer three-fourths of the right frontal sinus.

cases of osteoma of the maxillary sinus. At that time he added two cases of his own and in 1940[5] reported an additional case, making a total of 30 cases. Osteoma of the sphenoid sinuses is extremely rare; in fact, Hallberg and Begley[6] reported in May, 1950, that they were unable to find a single case of proved osteoma originating in the sphenoid sinus.

As to the incidence of the tumor in relation to sex, most authors have agreed that osteoma of the frontal sinus occurs more frequently in the male than in the female. In Teed's series on the frontal sinus, the proportion was 172 to 93. Nine

cases of osteoma of the ethmoid sinus, of which six were male and three female, were included in the Hallberg-Begley series. According to Rawlins there has been no appreciable increased incidence in the male where the maxillary sinus has been involved.

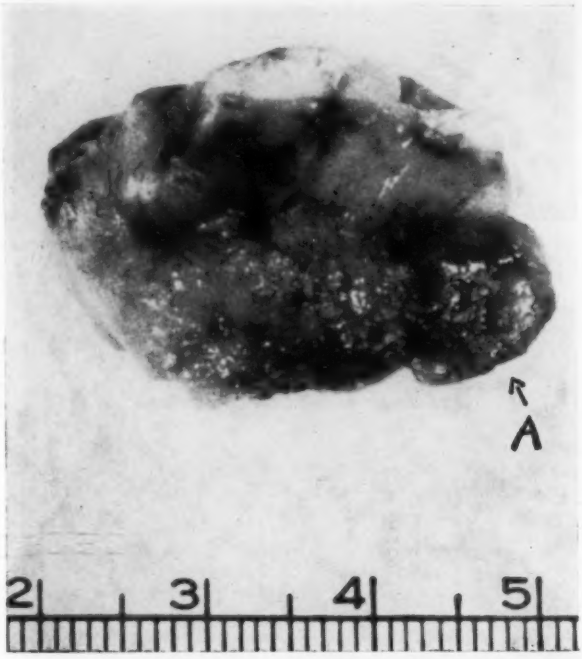

Fig. 2. Case 1. Photograph of osteoma. (A) Pedicle by which the tumor was attached to the anterior wall of the sinus at the lateral angle. It can be noted grossly that the pedicle is composed of spongy bone while the remaining surface of the tumor consists of dense bone.

Age distribution has been pointed out as one of the outstanding clinical features of osteomas. According to Armitage,[7] the tumor occurs in adolescence in 50 per cent of cases and before the age of 50 in 80 per cent. Among the cases reported and tabulated by Carmody,[8] the age range was from 11 to 61 years.

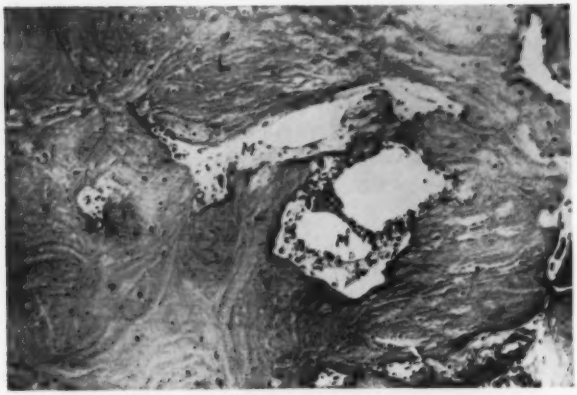

Fig. 3. Case 1. Photomicrograph (low power). (L) Lamellar bone; (M) marrow spaces filled with loose connective tissue and capillaries; (O) osteoblasts.

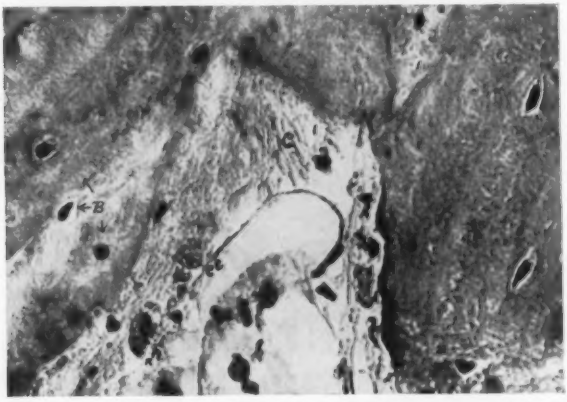

Fig. 4. Case 1. Photomicrograph (×400). (B) Bone corpuscles arranged without definite order; (C) loose connective tissue in marrow space; (cc) capillary within narrow space; (h) histiocyte.

ETIOLOGY.

Although many theories have been advanced, the exact etiology of this tumor is unknown. Some writers have stressed the importance of trauma; others have advanced the theory

that repeated infections may be a factor. According to Fetissof,[9] the two most popular theories have been: *1.* development from embryonal cartilage and *2.* development from embryonal periosteum. Fetissof's work, which agrees with that of Pfeiffer (1912) and Eckert (1922), is the most convincing. From a histological study of four cases, he concluded:

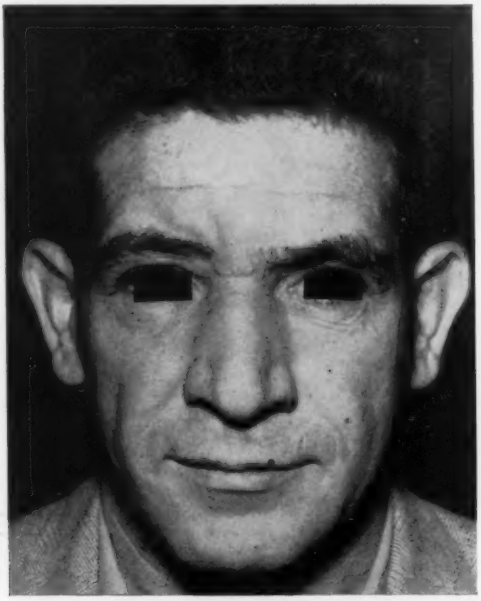

Fig. 5. Case 1. Photograph two months following operation.

1. The growth of the osteoma takes place from the inside outward by the metaplasia of fibrous tissue into bone tissue.

2. The osteoblastic ossificatory process that takes place at the periphery of the osteoma plays a secondary rôle in the growth of the tumor.

3. The fibrous tissue filling the interstices of spongiose bone is the direct continuation of the periosteum covering the osteoma from the outside.

4. The theory of origin of osteomas from the periosteum, split off at the stage of development of the organism of the periosteum, is the most fundamental.

Brunner and Spiesman[10] do not agree with Fetissof's theory that the spongiose portion of the osteoma is the more recently formed and that the compact bone represents an older part of the tumor. They state that in the expanding osteoma there is a slow but continual transformation of the compacta as well as the spongiosa and for that reason it is impossible to state

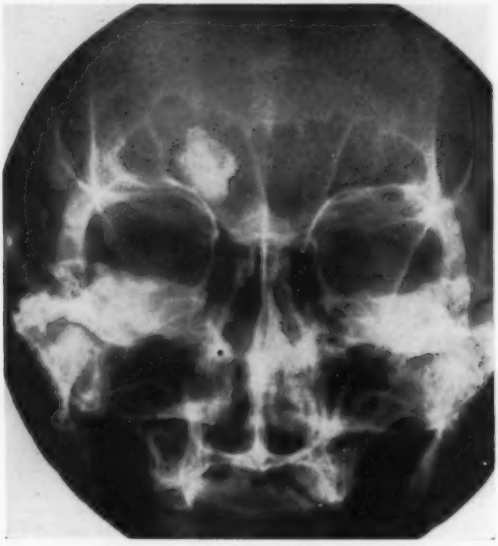

Fig. 6. Case 2. Photograph of X-rays showing the osteoma located in the middle third of the right frontal sinus.

which bone was formed first. They feel that the dense bone or solid cortex can also be deposited by the activity of the periosteum which forms the periosteal compacta. In other words, they apparently believe that the osteoblastic activity of the periosteum or submucosal periosteum at the periphery of the tumor plays a more important rôle in the bone formation

or expansion of the tumor. Fetissof indicated that this took place but that it was of minor importance, that the main increase in size of the osteoma was through metaplasia of the connective tissue within the trabeculae of the spongiose portion which penetrated the tumor from the periosteum.

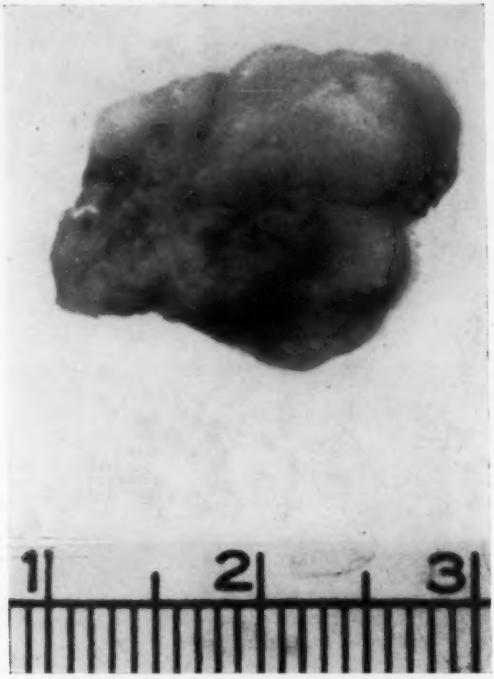

Fig. 7. Case 2. Photograph of osteoma.

SYMPTOMS AND COMPLICATIONS.

The symptoms produced by osteoma of the paranasal sinuses depend upon the cavity involved and the size and extension of the tumor. The tumor is asymptomatic unless basal structures are pressed on or ventilation or drainage of the sinus involved is interfered with. In fact, the diagnosis

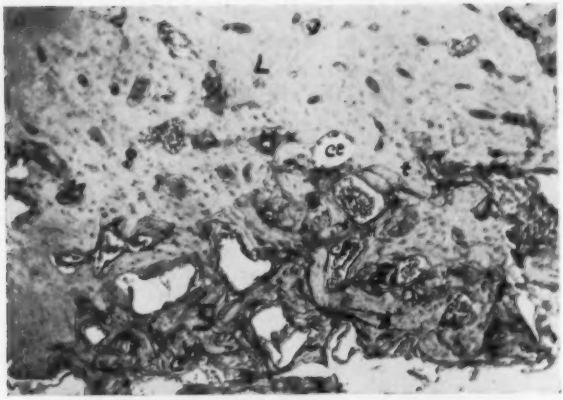

Fig. 8. Case 2. Low power photomicrograph of osteoma. (C) Connective tissue giving rise to osteoblastic ossification; (cc) capillaries within marrow spaces; (L) lamellar bone; (M) partially obliterated marrow spaces; (t) osteoid substance; (S) spongy bone with trabeculae.

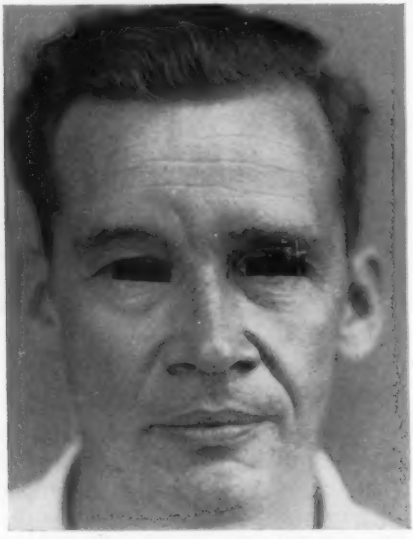

Fig. 9. Case 2. Photograph three months following operation.

is most commonly made as a result of taking routine X-rays of the sinuses. To illustrate, Childrey[11] has reported that 15 cases were noted in a review of 3,510 Roentgenograms of the sinuses, or an incidence of 0.43 per cent.

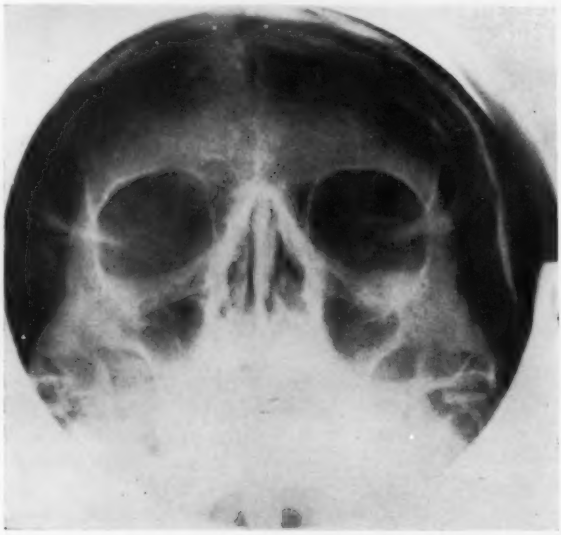

Fig. 10. Case 3. Photograph of X-rays of the sinuses showing the osteoma in the left infraorbital area.

The classical symptoms of osteoma of the frontal sinus are dizziness, headache and deformity; however, ocular symptoms are not uncommon when the anterior wall and floor of the sinus are eroded. When such invasion of the orbit occurs, unilateral nonpulsating exophthalmos usually results with the globe depressed forward, downward and outward. Periorbital swelling, proptosis, diplopia and impaired vision are associated symptoms. Supraorbital or nasociliar neuralgia may also result.[12] If the direction of the growth is inward, the inner table is eroded, and the lesion becomes intracranial with resultant complications such as meningitis, brain abscess, or pneumocephalus. The latter may or may not be associated

with cerebrospinal rhinorrhea. Growth into the region of the nasofrontal duct may isolate the sinus with the production of a mucocele or pyocele. In the series of 40 cases of osteoma of the frontal sinus prepared by Hallberg-Begley,[6] seven patients developed an infection of the frontal sinus on the involved side with resulting nasal discharge.

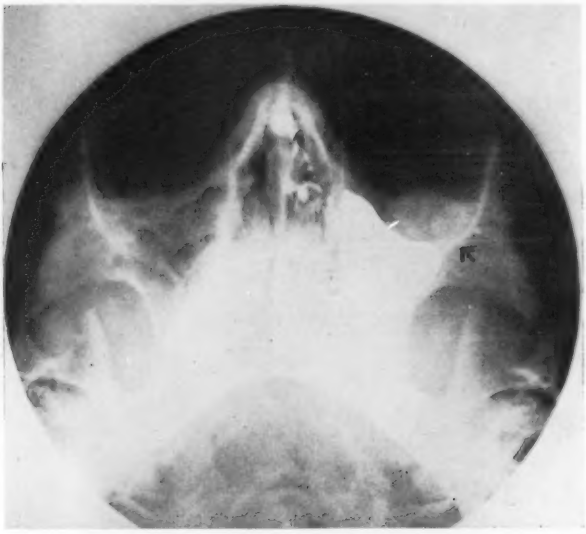

Fig. 11. Case 3. Photograph of X-rays of the sinuses showing lipiodal in the left maxillary sinus and the osteoma arising from the left maxillary bone.

Because of its restricted area, the ethmoid sinus produces symptoms earlier when attacked by osteoma than does the frontal sinus. Nasal obstruction and profuse nasal discharge on the involved side may be the earliest symptoms; however, the commonest symptom is frontal or maxillary pain. Again, orbital displacement is a symptom; but, in the case of ethmoidal osteoma, the globe usually is pushed forward and outward only, not downward as in the case of frontal involvement. Infection of the sinus is a complication. Cushing[13] has

Fig. 12. Case 3. Photograph of gross specimen. (P) Pedicle.

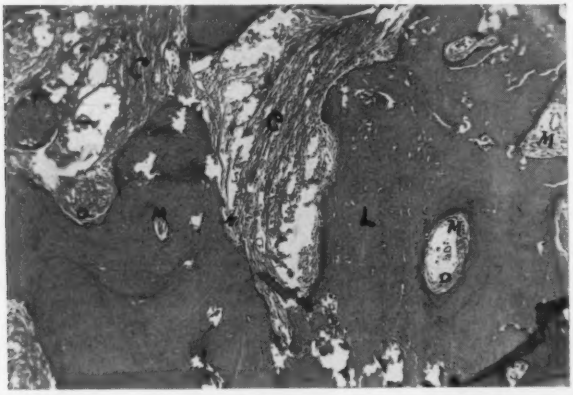

Fig. 13. Case 3. Low power photomicrograph of the osteoma. (C) Connective tissue, some of which is giving rise to bone by metaplasia; (H) Howship's lacuna; (L) lamellar bone containing bone corpuscles arranged without definite form; (M) marrow spaces containing connective tissue, capillaries and osteoblasts; (O) osteoblasts.

pointed out that perhaps the majority of ethmoidal osteomata ultimately lead to intracranial complications of one sort or another. He reported three such cases, in one of which cerebrospinal rhinorrea was present due to communication with the cerebral ventricle. In a second case, a large intracerebral pneumatocele was demonstrated; and a large intradural mucocele was noted in the third case.

Fig. 14. Case 3. Photograph showing a prominence in the left infraorbital area caused by the osteoma.

As in all cases of osteoma of the paranasal sinuses, the symptoms produced by maxillary involvement depend on the direction and extent of the pressure exerted by the tumor.

When it presses on the infraorbital nerve, neuralgic pain results. Pressure causing obstruction to the tear duct produces epiphora. Astigmatism and diplopia may be present from pressure on the eyeball; and pressure on the optic nerve may bring about impaired vision. Symptoms of deformity

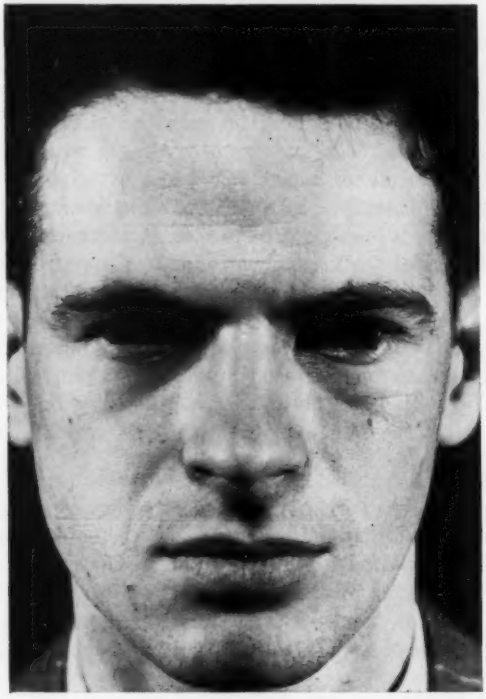

Fig. 15. Case 3. Photograph taken three months after operation.

may take the form of swelling of the cheek, deflection of the nasal wall toward the septum, downward displacement of the hard palate, and upward, outward displacement of the eyeball. Exophthalmos often occurs. Suppuration of the antrum is common; mucocele is rare.

HISTOPATHOLOGY.

Osteoma is the term applied to tumors composed of bone and, as Ewing[14] pointed out, should be limited to true neoplasms. These tumors vary in size from a few grams to 440 grams, the latter occurring in a case reported by Hilton[15] in 1836.

The bones of the face, in addition to giving rise to true osteomas, also are the seat of a variety of bony overgrowths which may be difficult to classify. Osteomas should be differentiated from exostoses or circumscribed masses projecting above the bony surface and from exostoses which lie within compact or cancellous bone. From the standpoint of histopathology, one is unable, as a rule, to make such a differentiation; therefore, it is important to identify each on the basis of gross and clinical features since the osteoma represents a neoplasm and the capacity to destroy adjacent structures by expansion. Exostoses usually arise from an overgrowth of subperiosteal bone on the basis of some inflammatory change in the periosteum and represent hyperplastic bony phenomena. Some of the hyperplasias of the maxilla may be very easily confused with true osteomas of the maxilla or with those arising within the maxillary sinus.

Microscopically, three types of osteomas are described: *1.* osteomas made up of eburnated bone (osteoma eburneum), *2.* spongiose osteoma (osteoma spongiosum), and *3.* mixed osteomas (osteoma mixtum). The mixed-type osteomas are the most common; and, in fact, in most osteomas the periphery is made up of compact bone and the center is composed of spongiose.

Osteomas originating within the sinus cavities are covered with pseudostratified columnar ciliated epithelium beneath which is found the submucosa and periosteum. Brunner and Spiesman[15] stated that in their cases the microscopic findings indicated that the periosteal compacta or lamellar bone at the surface of the tumor was laid down by the paranasal mucosa which serves as a periosteum.

The microscopic picture of osteomas in general differs from that of normal bone in that in the compact areas dense lamellated bone with few or no Haversian canals is seen. The lamellae are arranged in layers parallel to the surface. The bony corpuscles are arranged without definite order. The majority of the vascular canals run parallel to the long axis of the tumor. In the spongy portions there are irregular groups of interlacing trabeculae surrounded by connective tissue growing into the bony structure. The connective tissue within the trabeculae contains a great number of fibres between which fibroblasts may be seen. As indicated by the work of Fetissof,[9] it is by metaplasia from such connective tissue that the formation of new portions of bone occurs.

The osteoma's pedicle is usually composed of spongy bone and is penetrated by the periosteum which may run the entire thickness of the tumor and pass into the connective tissue filling the interstices of the trabeculae.

TREATMENT.

Surgery is, undoubtedly, the only treatment for osteoma of the paranasal sinuses; however, as late as 1918, Mackenty (quoted by Novick[16]) considered an osteoma inoperable. Today, with our improved surgical technique and antimicrobial therapy, the tumors should no longer be left alone until they have invaded or displaced neighboring structures or caused complications which may endanger life. Joseph Beck[17] stated that 48 per cent of unoperated cases of osteoma would die of complications arising from the presence of the tumor. Although this figure has been labeled "unsupported" by some, the fact that surgery is the only treatment has not been altered.

There has been some divergence of opinion as to when surgical intervention should be undertaken. It is the author's belief that once an osteoma has been demonstrated by X-ray and there is evidence of active growth clinically or by X-ray examination, the tumor should be removed. If the lesion is small and its location is such that moderate growth does not

threaten adjacent structures, then the growth of the lesion may be followed by X-ray examination at six month to one-year intervals. The case of a patient, age 15, reported by Coates and Krauss[18] demonstrates the rapidity with which these tumors can increase in size, particularly in the younger age group. Where possible, osteomas of the frontal sinus should be removed before the nasofrontal duct has been involved. To avoid intracranial complications, osteomas of the ethmoid sinus probably should be removed much earlier than those involving the frontal or maxillary sinuses.

In dealing with osteomas, one should remove not only the tumor but also its pedicle in order to avoid recurrence. Where the pedicle is attached to the posterior wall of the frontal sinus, it may be necessary to remove the posterior plate of the sinus; even so, there is little real danger and the plate will regenerate as it did in one of the cases reported by Coates and Krauss.[18]

Exposure of the osteoma may be accomplished by various incisions and approaches. For most of the frontal osteomas and for small lesions of the ethmoid sinus, various modifications of the Killian incision are most suitable. In the case of large osteomas of the orbitoethmoidal type with intracranial extension, with or without complications, it may be necessary to employ the intracranial approach as advocated by Cushing[13] or a combination of trans-sinal and intracranial approach. Osteomas of the maxillary sinus may be approached by either the Caldwell-Luc or the Denker methods.

CASE REPORTS.

Case 1: L. R., a white male, aged 30, was admitted to the otolaryngological service of Halloran Veterans Hospital the latter part of March, 1948. The patient complained of severe right frontal and right temporal headache. The headaches were first noticed in 1942 when they would occur at three to five-week intervals and last for two to three days. They were less marked in 1944 and 1945. From December, 1946, to January, 1948, the headaches were moderately severe, recurred every three to six weeks and lasted one and one-half to three days. The patient stated that they were continuous from January until March, 1948. They would be quite severe for eight to nine days, would ease off for two or three days and then become severe again. The patient could neither sleep nor do a full day's work during the severe attacks.

Routine X-ray of the paranasal sinuses revealed a large osteoma of the right frontal sinus which filled the lateral three-fourths of the sinus.

On April 1, 1948, the patient was operated upon under local anesthesia. The anterior wall and floor of the right frontal sinus were exposed by an incision along the lower margin of the eyebrow. The floor of the frontal sinus was entered at its inner angle, using the perforating burr. The tumor completely filled the outer three-fourths of the sinus and could not be removed *in toto* without sacrificing most of the anterior wall and floor of the frontal sinus. The growth had almost completely eroded the anterior wall and floor of the right frontal sinus in the area of the supraorbital foramen. The osteoma was attached by a small friable pedicle to the anterior wall at the lateral angle of the sinus. The pedicle and adjacent bone were removed. The posterior wall of the sinus was intact and covered by normal mucous membrane. The frontal ostium was not involved and was left untouched. There was normal mucous membrane about the frontal ostium for at least 1 cm. A small catheter drain was inserted at the outer angle and the wound closed in the routine manner. A pressure dressing was applied.

Twenty-four hours later the sinus was irrigated with penicillin solution colored with methylene blue. The solution freely entered the nose and the drain was removed. The wound healed per primam. Postoperatively the only subjective complaint was slight diplopia which soon disappeared.

The pathology report by Dr. Oscar Auerbach follows:

Macroscopic: Present is specimen of bone measuring 3 by 2 by 1.5 cm. The surfaces are lobulated and the lobules vary from 2 mm. to 7 mm. The surface is pinkish white and smooth for the most part, but one surface is rough. On section, the tissue is stony hard. The cut surface is white to pinkish white and the surface is rough.

Microscopic: Microscopic examination shows thick and irregular bony trabeculae with narrow marrow spaces. Within the marrow spaces are fibroblasts, histiocytes and some capillaries.

Diagnosis: Osteoma.

Case 2: E. P. C., a white male, aged 38, was admitted to the otolaryngological service of Halloran Veterans Hospital in May, 1948, with the chief complaint of right-sided headaches.

The patient gave a history of generalized headaches occurring from the age of 10 until the age of 17, at which time they disappeared. He again developed headaches in 1943 at the age of 29. At this time they were localized about the right eye with a tendency to radiate back to the right occipital area. At times there was a throbbing sensation in the right temporal area and about the right eye which would keep the patient from going to sleep. The headaches increased in severity until he was unable to work.

Routine X-rays of the sinuses revealed a large osteoma of the right frontal sinus measuring 12 by 15 mm. in the anterior-posterior projection and 1 cm. in the lateral projection.

On May 6, 1948, the patient was operated upon under local anesthesia. The incision was made along the lower margin of the right eyebrow. The floor of the frontal sinus was entered at its angle, using the per-

forating burr. The anterior wall of the frontal sinus was removed around the osteoma, using a small Kerrison rongeur. The tumor filled about one-half of the frontal sinus but did not encroach upon the frontal ostium. It was atta ched to the middle third of the posterior wall of the sinus by a very small pedicle. No destruction of adjacent bone was noted. The osteoma was easily freed and the base of the pedicle, which measured only about 3 to 4 mm. across, was destroyed with a polishing burr. The mucous membrane about the frontal ostium was normal and it was left intact as far out as possible, which was over 1 cm. A small catheter drain was inserted at the outer angle of the wound and the wound closed in the routine manner. A pressure dressing was applied.

Postoperatively the sinus was irrigated daily through the catheter drain with penicillin solution. The solution entered the nose freely on the third postoperative day and the drain was removed the following day. The patient developed a stitch abscess at the inner angle of the wound, which was easily drained and which did not involve the sinus. The convalescence was otherwise uneventful. Although the patient still experiences an occasional headache, he states that they are not of the same type or severity.

The pathology report by Dr. Oscar Auerbach follows:

Macroscopic: Present is a portion of lobulated bone measuring 2 by 1.5 by 1 cm. The surface for the most part is yellow-gray in color and smooth and in one area, 0.5 cm. in diameter, the surface is red-gray and roughened.

Microscopic: Microscopic examination shows thick and irregular calcified bony trabeculae with, for the most part, narrow marrow spaces. The spaces contain pink, dense connective tissue and the usual marrow cells are absent.

Diagnosis: Osteoma of frontal sinus.

Case 3: H. L., a white male, aged 24, was admitted to the otolaryngological service of Halloran Veterans Hospital, Dec. 15, 1949. The patient had complained of a "stinging feeling" in his left cheek for two months prior to admission. He had noticed a hard lump under his left eye which had increased in size during the past two and one-half years.

Routine X-rays of the sinuses and X-rays of the sinuses with lipiodal instilled into the left maxillary sinus showed a bony tumor over the left maxillary bone at the junction of the maxillary and zygomatic bones. The tumor did not appear to enter the maxillary sinus.

The local examination of the ears, nose and throat and the general physical examination were negative.

On Dec. 21, 1949, the patient was operated upon under local anesthesia. The osteoma was exposed through a small horizontal skin incision 1 cm. below the left infraorbital rim. The incision was carried through the soft tissues and the tumor was exposed lying in a shallow trough on the outer upper aspect of the maxillary bone. The osteoma was easily removed by introducing a curved gouge along the cleavage line found between the osteoma and its bed in the maxillary bone. The osteoma was attached by a definite pedicle at the suture line between the maxillary and zygomatic bones. The base of the pedicle was destroyed with the electric polishing burr. The skin was closed with a single subcuticular dermal suture.

The pathology report by Dr. Oscar Auerbach follows:

Macroscopic: Present are two portions of bony tissue, the largest of which measures 2.1 by 1.4 by 1 cm. and is reniform in shape. The smaller portion of bony tissue measures 1 by 0.5 by 0.5 cm. and is rounded in one aspect and irregular on the opposite aspect. The surface of the larger portion is composed of dense pink-white bone which in some aspects is smooth while in other aspects it is slightly roughened.

Microscopic: Microscopic examination shows a portion of bony tissue composed of wide, dense trabeculae. These trabeculae vary greatly in thickness from segment to segment. In areas the mosaic lines are prominent. Some of the marrow spaces contain dense bands of connective tissue consisting of fibroblasts and capillaries while others are filled with fat.

Diagnosis: Osteoma, left infraorbital region.

COMMENTS.

Three cases of osteomas of the facial bones are reported. All three cases occurred in males between the ages of 24 and 38 years. In the first two cases the osteomas definitely arose from within the right frontal sinus, the pedicle arising from the anterior wall in the first case and from the posterior plate in the second. In the third case the pedicle was attached to the outer surface of the left maxilla in the infraorbital area near the suture line between the maxillary and zygomatic bones. The tumor did not enter the cavity of the maxillary sinus and must be classified as an osteoma of the maxilla rather than of the maxillary sinus.

There was evidence of active growth and expansion of the osteoma in at least two of the cases. In the first case the tumor had almost eroded through the anterior wall and floor of the right frontal sinus and was giving rise to frontal headache which was presumed to be supraorbital nerve neuralgia and secondary to pressure from the tumor. Case 3 revealed clinical evidence of appreciable growth of the osteoma during the two and one-half years prior to operation. Although the tumor had attained considerable growth in the second case, we had no definite clinical or X-ray evidence to indicate the rapidity of growth; but due to the proximity of the tumor to the nasofrontal duct, it was deemed wise to remove the growth before further encroachment upon this area occurred.

In the two cases involving the right frontal sinus the nasofrontal duct had not been involved and the mucous membrane of the sinuses in this area was left intact and the nasofrontal duct was not disturbed. This allowed the mucous membrane to regenerate in a healthy functioning sinus. It is obvious that both cases were ideal for this form of treatment. Where osteomas of the frontal sinus are not diagnosed until they have involved the nasofrontal duct or, while under observation, are allowed to encroach on this area by growth, the incidence of complications is greatly increased. Even if the tumor is completely removed by the Lynch-type frontoethmoid operation where the nasofrontal duct is necessarily altered, the chances of a mucocele or pyocele developing within a period of 10 to 12 years are quite good.

The removal of the osteoma in the third case presented no real problem and the cosmetic result was reasonably good.

Grossly and microscopically, the three osteomas reported here showed evidence of both dense, lamellar bone and spongy type bone, the pedicles consisting primarily of spongy bone and the peripheral portions of the more dense lamellar bone; hence, the three would be classified as mixed osteomas. A typical section showing both dense osteoid bone and spongy bone is shown in Fig. 8, Case 2.

Bone formation appeared to be derived primarily from metaplasia of the connective tissue penetrating the trabeculae from the pedicle (see Fig. 13, Case 3). Some bone formation probably occurred from the periosteum at the periphery of the tumor by an osteoblastic ossificatory process; however, no definite observations could be made on this point due to the limited material which was sectioned by the technician from each tumor.

CONCLUSIONS.

1. Osteoma of the paranasal sinuses is a fairly uncommon tumor. The frontal sinus is most frequently involved.

2. Fetissof's theory of origin of osteomas from the periosteum is the theory most acceptable.

3. Patients having symptoms suggesting osteoma of the paranasal sinuses should have routine X-rays of the sinuses.

4. Evidence of an increase in the size of an osteoma sufficient to threaten adjacent structures is an indication for surgical removal.

5. Surgery is the only treatment for these lesions, and care should be exercised to remove not only the tumor but also the pedicle to avoid recurrence.

REFERENCES.

1. Dahman (cited by *Fetissof*, A. G.): Pathogenesis of Osteoma of Nasal Accessory Sinuses. *Ann. Otol., Rhinol. and Laryngol.*, 38:404, June, 1929.

2. Malan (cited by Salvadori, G.): Osteoma of the Frontal Sinus. *Arch. Otolaryngol.*, 35:671, Apr., 1942.

3. Teed, R. W.: Primary Osteoma of the Frontal Sinus. *Arch. Otolaryngol.*, 33:255, Feb., 1941.

4. Rawlins, A. G.: Osteoma of the Maxillary Sinus. *Ann. Otol., Rhinol. and Laryngol.*, 47:735, Sept., 1938.

5. Rawlins, A. G.: Osteoma of the Maxillary Sinus: A Case. *Arch. Otolaryngol.*, 32:642, Oct., 1940.

6. Hallberg, O. S., and Begley, J. W.: Origin and Treatment of Osteomas of the Paranasal Sinuses. *Arch. Otolaryngol.*, 51:750, May, 1950.

7. Armitage, G.: Osteoma of the Frontal Sinus; with Particular Reference to Its Intracranial Complications and with Report of a Case. *Brit. Jour. Surg.*, 18:565, 1930-1931.

8. Carmody, T. E.: Osteoma of the Nasal Accessory Sinuses. *Ann. Otol., Rhinol. and Laryngol.*, 44:626, Sept., 1935.

9. Fetissof, A. G.: Pathogenesis of Osteoma of Nasal Accessory Sinuses. *Ann. Otol., Rhinol. and Laryngol.*, 38:404, June, 1929.

10. Brunner, H., and Spiesman, I. G.: Osteoma of the Frontal and Ethmoid Sinuses. *Ann. Otol., Rhinol. and Laryngol.*, 57:714, Sept., 1948.

11. Childrey, J. H.: Osteoma of the Sinuses, Frontal and Sphenoid Bone. *Arch. Otolaryngol.*, 30:63, July, 1939.

12. Fenton, R. A.: Osteoma Causing Nasociliary Neuralgia. *Ann. Otol., Rhinol. and Laryngol.*, 42:911, Sept., 1933.

13. Cushing, H.: Experiences with Orbitoethmoidal Osteomata Having Intracranial Complications. *Surg., Gynec. and Obst.*, 44:721, June, 1927.

14. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors. Ed. 3. Philadelpha, Pa.: W. B. Saunders Co., 1928.

15. Hilton: Case of a Large Bony Tumor in the Face Completely Removed by Spontaneous Separation. *Guy's Hosp. Rep.*, 1:493, 1836.

16. Novick, J. N.: Osteoma of the Frontal Sinuses. *Arch. Otolaryngol.*, 46:655, Nov., 1947.

17. Beck, J. C.: Some Uncommon Types of Neoplasms About the Head and Neck. The Laryngoscope, 40:511, July, 1930.

18. Coates, G. M., and Krauss, F.: Osteoma of the Frontal Sinus. *Ann. Otol., Rhinol. and Laryngol.*, 50:450, June, 1941.

RECRUITMENT OF LOUDNESS IN THE DIFFERENTIAL DIAGNOSIS OF END-ORGAN AND NERVE FIBRE DEAFNESS.*

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In 1936, Fowler[5] described a method of testing hearing which was said to differentiate neural from conduction deafness without recourse to determinations of the thresholds of either bone conduction or air conduction of sound. This test consists of the measurement of the recruitment of loudness with increasing intensity of sound. Fowler first demonstrated the presence of recruitment by balancing the loudness of a sound which was alternately thrown first into one ear and then into the other. This was referred to as the "alternate binaural loudness balance" test.

Fowler noted varying differences in loudness necessary for balance in various types of deafness. He found that the relative sensation of loudness did not change in the two ears at any intensity of sound in conduction deafness. On the other hand, he observed in some cases of nerve deafness that the ear with the poorer hearing could hear pure tones more and more distinctly as the intensity was increased. In other words, loudness recruitment was present. "Nerve deafness" was used by Fowler in the broadest sense and included lesions of the organ of Corti as well as of the nerve fibres and ganglion cells.

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†Abstract of thesis submitted by Dr. Eby to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Otolaryngology and Rhinology.

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The phenomenon of the recovery of relative capacity to hear at higher levels of intensity by an ear which fails to hear at threshold of intensity is referred to as the loudness recruitment phenomenon.[13]

When Dix, Hallpike and Hood,[5] in 1948, observed that the loudness recruitment phenomenon was characteristically present in Ménière's disease and absent in cases of VIIIth nerve tumors and lesions at the cerebellopontine angle, they gave new importance to recruitment tests in the differential diagnosis of lesions of the end-organ and nerve fibres which produce deafness.

The same authors,[6] one year later, reported that further observations in two cases of Ménière's disease and two cases of neurofibromata of the VIIIth nerve revealed differences in the intelligibility of speech associated with the two lesions. They found that, in Ménière's disease there was a recruitment of loudness but not recruitment of speech intelligibility; on the other hand, in the presence of neurofibromata, speech became more intelligible with increasing amplification up to a point. These findings sharply contradicted previous concepts as to the physiologic mechanism of recruitment. For this reason confirmation of the hypothesis that recruitment of loudness is present in a lesion of the epithelium of the end-organ, whereas recruitment of loudness is not present in a lesion of the nerve fibres or ganglion cells of the VIIIth nerve, was considered necessary.

In our study, alternate binaural loudness balance tests were performed on 32 patients with unilateral deafness diagnosed as Ménière's disease (end-organ lesion), on one patient with unilateral deafness thought to be the result of a vascular accident involving the organ of Corti, and on 16 patients with unilateral auditory nerve deafness due to a variety of causes.

In Table 1 is given the diagnosis in the 16 cases of nerve deafness in which alternate binaural loudness balance tests were made. The diagnosis of nerve fibre involvement was substantiated by the operative findings in eight cases. VIIIth

TABLE 1.

LOUDNESS RECRUITMENT AND DIPLACUSIS BINAURALIS DYSHARMONICA IN 16 CASES OF NERVE FIBRE DEAFNESS.

Case	Diagnosis	Diplacusis	Loudness Recruitment
1	Neurofibroma of right acoustic nerve	—	—
2	Meningioma at right cerebellopontine angle	Present	—
3	Osteoma at right cerebellopontine angle	—	—
4	Neurofibroma of right acoustic nerve	—	—
5	Meningioma at left cerebellopontine angle	—	—
6	Astrocytoma of left temporoparietal lobe	—	—
7	Left intracerebral hematoma, secondary to arteriovenous fistula or angioma	—	—
8	Neurofibroma of right acoustic nerve	—	—
9	Paget's disease of the skull	—	—
10	Pneumococcic meningitis	—	—
11	Meningitis	—	—
12	Thrombosis of anterior inferior cerebellar or basilar artery	—	Incomplete
13	Probable brain tumor with resulting VIIIth nerve damage	—	—
14	Possible VIIIth nerve tumor	—	Incomplete
15	Vascular accident involving VIIIth nerve fibres	—	—
16	Parkinson's disease, cochlear nerve damage due to disturbed blood supply	—	Incomplete

nerve tumors and lesions in the cerebellopontine angle were found to be present. In the eight other cases in which the VIIIth nerve fibres were presumed to be affected but operation was not performed, the diagnosis was based on Roentgenologic, neurologic and otologic findings.

Speech intelligibility tests also were made on both groups of patients whenever such tests were possible.

EQUIPMENT AND TESTING METHODS.

The routine audiometric tests were made on an Audio-Development Co. Audiometer, Model 50E, by an experienced technician in a quiet room.

Alternate binaural loudness balance and speech intelligibility tests were carried out in a sound insulated room in which there was a noise level of approximately 35 db when tested with a general radio sound level meter. These tests were made with a Maico E1 Audiometer. Thresholds for speech reception were determined by use of the spondee word test developed by Hudgins and his co-workers. Speech intelligibility was determined by use of the phonetically balanced (P. B.) word lists worked out by Egan and his group at the Psycho-Acoustic Laboratory of Harvard University.

When speech intelligibility tests were made, the microphone was held 2 inches (5.08 cm.) from the examiner's lips. The intensity of the voice and inflection were maintained as constant as possible with the assistance of a speech monitoring meter (Maico Part No. 4506). With the meter adjusted to the test conditions, the voice was controlled in order to cause a swing of the meter pointer to the 0 db point on the carrying phase preceding each word. Then each word of the phonetically balanced (P. B.) word list was allowed to vary according to its own phonetic composition.

The intensity level at which speech sounds became uncomfortable to the listener was taken as the threshold of discomfort for the ear.

RESULTS.

Loudness recruitment was absent on the alternate binaural loudness balance test in the eight cases of nerve deafness in which the diagnosis was made at operation and in all but three of the remaining eight cases of nerve lesions. In the three cases in which some evidence of recruitment was found, it was of only a slight degree and in no instance was recruitment complete.

In 15 of the 16 cases of nerve fibre deafness, diplacusis binauralis dysharmonica was absent.

In Fig. 1 are shown an audiogram and the results of the alternate binaural loudness balance tests on a patient who had

a neurofibroma of the VIIIth nerve. The absence of loudness recruitment is indicated by the almost parallel loudness balance lines joining the sound intensities which produced equal loudness in the two ears. Audiograms in other cases were similar (see Figs. 2 and 3).

The results of alternate binaural loudness balance tests on 33 patients whose deafness was presumed to be due to lesions of the end-organ of hearing are given in Table 2. Loudness

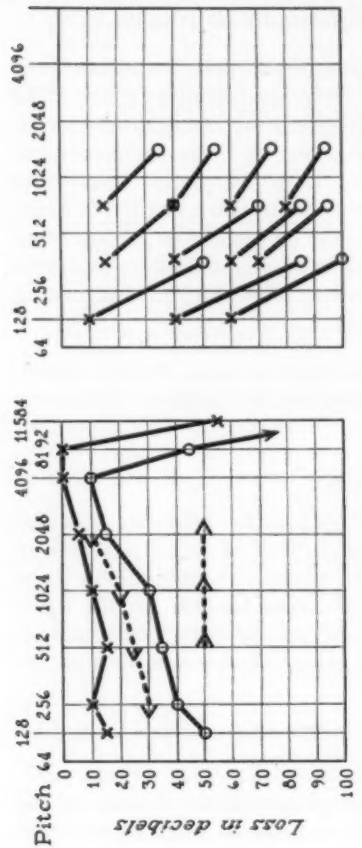

Fig. 1. Case 1, Table 1. Audiogram in a case of neurofibroma of the VIIIth nerve giving results of the alternate binaural loudness balance and speech intelligibility tests.

recruitment was present in every case. In only two cases was recruitment found to be incomplete within the limits of the audiometric scale. In 17 of the 33 cases, over-recruitment was expressed on the test. When a tone is heard by the defective ear with loudness equal to that heard in the normal ear even though the intensity of sound delivered to the defective ear is less, over-recruitment is said to be present.

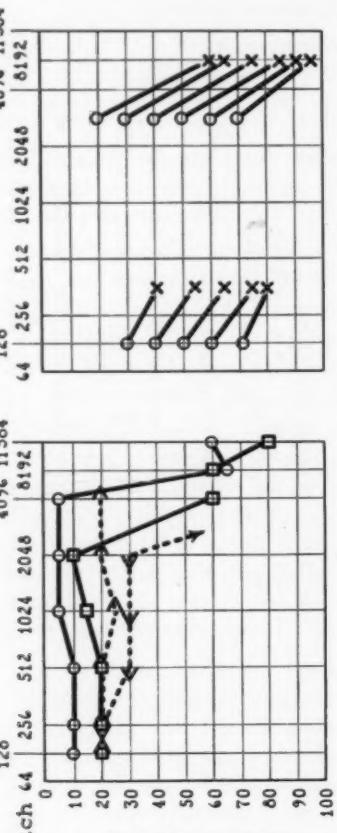

Fig. 2. Case 6, Table 1. Audiogram in a case of astrocytoma of the temporoparietal lobe producing pressure on the VIIIth nerve. Results of the alternate binaural loudness balance and speech intelligibility tests are shown.

Twenty-seven of the 33 patients were tested for diplacusis binauralis dysharmonica. It was present in 23. Diplacusis binauralis dysharmonica is usually associated with a disorder of the end-organ of hearing.

An audiogram and the results of the alternate binaural loudness balance tests in a case of Ménière's disease are shown in Fig. 2. The presence of loudness recruitment is shown by

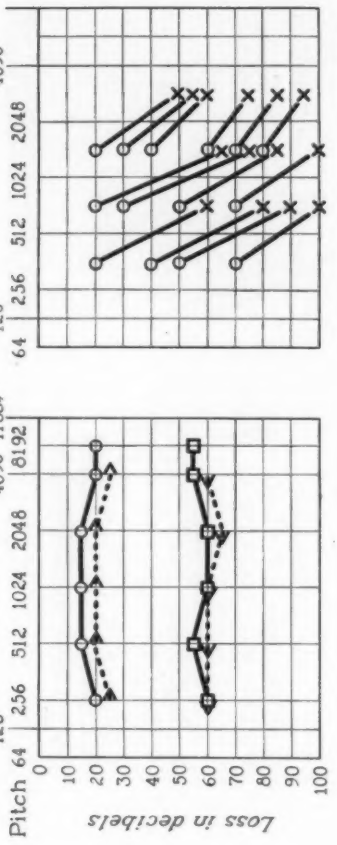

FIG. 3. Case 7, Table 1. Audiograms in a case of a hematoma in the left temporoparietal lobe secondary to an arteriovenous fistula or angioma. Results of the alternate binaural loudness and speech intelligibility tests are shown.

TABLE 2.

SUMMARY OF FINDINGS OF LOUDNESS RECRUITMENT AND DIPLACUSIS BINAURALIS DYSHARMONICA IN CASES OF END-ORGAN DEAFNESS.

Cases		Total Cases
Loudness recruitment		33
Incomplete	2	
Complete	31	
Over-recruitment	17	
Diplacusis		27
Present	23	
Absent	4	

the loudness balance lines which become shorter and more horizontal with each increase of intensity until complete recruitment is reached.

Speech Tests.—In six cases of nerve fibre deafness speech intelligibility was tested by use of phonetically balanced (P. B.) word lists. In five of these cases speech became more and more intelligible as the intensity of sound was increased until the limit of the audiometric scale was reached. An example of such observations in a case of VIIIth nerve neurofibroma is shown in Fig. 1.

In 15 of the 17 cases in which a lesion of the end-organ of hearing was presumed to be present and these tests were made, speech intelligibility reached a maximum, then declined as the intensity of sound was increased further within the limits of intensity of the audiometer. An example of this observation in a representative case of Ménière's disease is shown (see Fig. 4).

The dynamic range for speech was narrowed in cases of end-organ deafness. In 19 cases the average difference between the threshold for speech reception and the most comfortable listening level was 18.42 db. This finding was in contrast to the wider span between these two levels found in seven cases of nerve fibre deafness in which the span was determined. In these cases the average difference was 28.57 db.

The threshold of discomfort for speech was determined in 12 cases of Ménière's disease and in one case in which deafness was thought to be due to a lesion in the organ of Corti. Of this group, the threshold for discomfort was found to be relatively lower than that found in the seven cases of nerve fibre deafness in which the test was made. In seven cases of nerve fibre deafness the threshold of discomfort was found to be beyond the limit of the audiometric scale.

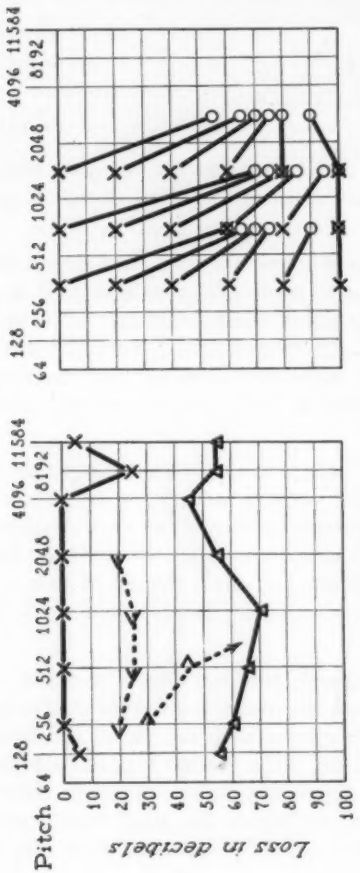

Fig. 4. Audiogram in a case of Ménière's disease with the results of alternate binaural loudness balance and speech intelligibility tests.

COMMENT.

In a discussion given on the recruitment of loudness phenomenon at San Francisco, in 1950, Fowler[9] differed sharply with the conclusion of Dix, Hallpike and Hood.[5] He stated that in performing loudness balance tests, if one ear has normal hearing and the other is deafened by a neural or so-called nerve deafness, the difference between the two ears at threshold will disappear as the sound is increased over threshold until at high intensities the sound appears just as loud, or sometimes even louder, to the deafened ear as it does to the normal ear. This pathologic increase in the increment of loudness is called "recruitment of loudness" and is pathognomonic of auditory neural lesions.

In regard to the report of Dix, Hallpike and Hood, Fowler stated that their hypothesis that recruitment arises only in lesions of the end-organ and, therefore, its absence in neural deafness provided a test for the differentiation between end-organ and neural degeneration deafness was interesting and he wished that it could be true. He, however, had never observed it unless there was insufficient hearing to permit the test. He offered as evidence an audiogram taken before operation in a case of neurofibroma of the VIIIth nerve in which recruitment was clearly shown.

Fowler also embraced the hypothesis of von Bekesy that stationary or increased difference limen arises from the same fundamental physiologic disorder as does recruitment of loudness. Our findings on testing patients with degenerative nerve lesions and presumed endolymphatic hydrops are diametrically opposed to the opinion expressed by Fowler and are in agreement with the findings of Dix, Hallpike and Hood.[5]

It is of great importance that this question be settled, because in the differential diagnosis of Ménière's disease, if one can have complete confidence in the differentiating value of the loudness balance test a forward step in the ease of differentiation of this disorder from conditions likely to be confused with it will have been taken.

In our experience at the Mayo Clinic we have not as yet been led astray by the loudness balance test. In one instance in which neurosurgical exploration of the cerebellopontine angle was done because of conflicting neurologic findings in the presence of loudness balance test showing complete recruitment no neural lesion was found and an intracranial division of the VIIIth cranial nerve relieved the symptoms.

Recent evidence presented tends to cast doubt on Fowler's assumption that an increased or unchanged difference limen and the recruitment of loudness phenomenon are different aspects of the same underlying physiologic change.

When the intensity of a sound is increased continuously, the corresponding sensation of loudness does not increase continuously but in discrete steps. A change in intensity sufficient to produce a just perceptible change of loudness is called "the intensity difference limen for loudness." It is well known that the normal intensity difference limen for loudness is relatively large at low intensities of sensation and much smaller at higher intensities of sensation. When recruitment is assumed to be present, the difference limen is much smaller than normal at low intensities and increases to normal values at higher intensities; therefore, if the difference limen increases or remains unchanged with increasing levels (intensities) of sensation, it is assumed that recruitment is present. If, on the other hand, the difference limen decreases with increasing levels of sensation, it is assumed that there is no recruitment.[4]

As found by Fowler,[9] von Bekesy and others, recruitment of loudness is common to both lesions of the epithelial end-organ and lesions of the nerve fibres and ganglion cells. Dix, Hallpike and Hood,[5] de Bruine-Altes, Mygind,[11,12] and now one of us (Eby), have found that recruitment of loudness is not present in nerve fibre and ganglion cell lesions. This indicates that there is some important difference in physiologic backgrounds of changes in difference limen and recruitment of loudness. It is for this reason that we believe Fowler's assumption to be incorrect.

Fowler's[9] theoretical explanation fits the conditions of change in difference limen perfectly, although as he pointed out it is unsatisfactory for explanation of the phenomenon of hyper-recruitment or over-recruitment.

Probably the hypothesis suggested by Bustamante Gurria and Garibay best explains the difference in sudden and slower changes in difference limen. Von Bekesy concluded from his investigation that reduction in the number of functioning nerve cells in a given area of the receptor organ of hearing would be indicated by an increasing of the difference limen. Mygind[12] considered von Bekesy's explanation completely acceptable for neural lesions. Mygind[11] accepted Shambaugh's suggestion that the tectorial membrane is the principal factor in analysis of tone. He pointed out that the nerve apparatus has been repeatedly found completely normal in disorders of hearing due to changes in the tectorial membrane; however, in changes in the tectorial membrane the distribution of the secondary sound waves on the organ of Corti show an unregulated pattern of discontinuous waves producing the same effect on the sensation of loudness as is produced by irregular degeneration of nerve fibres or ganglion cells.

Whatever the virtue, or lack of it, in Mygind's[11] theoretic explanation of the reason, change of difference limen can be found in both nerve deafness and end-organ deafness, the evidence submitted seems to be convincing that while diplacusis and recruitment are different aspects of the same underlying process, a stationary or decreased difference limen results from some different physical derangement.

We believe, therefore, that speech intelligibility tests, like the loudness balance tests, may be considered helpful in distinguishing end-organ from nerve fibre deafness.

SUMMARY AND CONCLUSIONS.

Alternate binaural loudness balance tests were made on 32 patients with unilateral deafness diagnosed as Ménière's disease, and on one patient with unilateral deafness thought to

be the result of a vascular accident which involved the organ of Corti. Loudness recruitment was present in every case. In 17 cases there was evidence of over-recruitment.

Alternate binaural loudness balance tests also were made on 16 patients with deafness due to degeneration of nerve fibres. In eight cases intracranial lesions which involved the VIIIth nerve were found at operation. In all eight, loudness recruitment was found to be absent. In eight other cases in which there was a probable lesion of VIIIth nerve fibres, there was little or no evidence of loudness recruitment.

These findings add further evidence to indicate that loudness recruitment is present in a lesion of the organ of Corti but absent in a lesion of the auditory nerve fibres.

Diplacusis binauralis dysharmonica was present in 23 of 27 cases of end-organ deafness in which this was tested, whereas it was present in only one out of 16 cases of nerve fibre deafness.

Speech intelligibility tests were made in 17 cases of deafness in which it can be presumed that a lesion of the end-organ was present. In 15 of these 17 cases, speech intelligibility reached a maximum, then declined with further increases of intensity within the intensity range of the audiometer.

Speech intelligibility tests were made in six cases of nerve fibre degeneration. In five of these cases, speech continued to become more and more intelligible with increased intensity of sound, until the limit of the audiometric scale was reached.

It would seem from these findings that discrimination deafness is an expression of the same fundamental disorder which is responsible for recruitment.

In 19 cases of end-organ deafness in which the speech reception threshold and most comfortable listening level were determined, the average difference in intensity of sound between these two levels was 18.42 db. In the seven cases of

nerve fibre deafness in which these levels were determined, there was a wider span between them, an average difference of 28.57 db.

The threshold of discomfort for speech was determined in 12 cases of deafness due to Ménière's disease and in one case of deafness due to a vascular accident involving the organ of Corti. In every case this threshold was found to be within the limits of the audiometric scale. In the seven cases of nerve fibre deafness in which an attempt was made to determine this threshold, it was found in each instance to be beyond the limit of the audiometric scale.

These findings, the early decline in the speech intelligibility, the decrease in the span of speech intelligibility, and the lower threshold of discomfort for speech in our cases of end-organ deafness seem to indicate the presence of loudness recruitment. In contrast, the results of speech tests in our series of cases of nerve fibre deafness seem to indicate the absence of loudness recruitment.

REFERENCES.

1. VON BEKESY, GEORG: The Recruitment Phenomenon and Difference Limen in Hearing and Vibration Sense. THE LARYNGOSCOPE, 57:765-777, Dec., 1947.

2. DE BRUINE-ALTES, JOHANNA C.: The Symptom of Regression in Different Kinds of Deafness. Gronigen: J. B. Wolters, 1946, 176 pp.

3. BUSTAMANTE GURRIA, ANDRES, and GARIBAY, ALFONSO: Comentarios al fenómeno de Fowler. *Prensa med. mex.*, 9:124-125, Aug. 15, 1944.

4. DENES, P., and NAUNTON, R. F.: The Clinical Detection of Auditory Recruitment. *Jour. Laryngol. and Otol.*, 64:375-398, July, 1950.

5. DIX, M. R.; HALLPIKE, C. S., and HOOD, J. D.: Observations Upon the Loudness Recruitment Phenomenon, with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and VIIIth Nerve. *Proc. Roy. Soc. Med.*, 41:516-526, Aug.; *Jour. Laryngol. and Otol.*, 62:671-686, Nov., 1948.

6. DIX, M. R.; HALLPIKE, C. S., and HOOD, J. D.: "Nerve" Deafness: Its Clinical Criteria, Old and New. *Proc. Roy. Soc. Med.*, 42:527-536, July, 1949.

7. EGAN, J. P.: Articulation Testing Methods. THE LARYNGOSCOPE, 58: 955-991, Sept., 1948.

8. Fowler, E. P.: A Method for the Early Detection of Otosclerosis; a Study of Sounds Well Above Threshold. *Arch. Otolaryngol.*, 24:731-741, Dec., 1936.

9. Fowler, E. P.: The Recruitment of Loudness Phenomenon. The Laryngoscope, 60:680-695, July, 1950.

10. Hudgins, C. V.; Hawkins, J. E.; Karlin, J. E., and Stevens, S. S.: The Development of Recorded Auditory Tests for Measuring Hearing Loss for Speech. The Laryngoscope, 57:57-89, Jan., 1947.

11. Mygind, S. H.: Further Labyrinthine Studies. II—On the Labyrinthine Transformation of the Acoustic Vibrations to Pitch-Differentiated Nervous Impulses. *Acta Oto-Laryngol.*, Supp. 68, pp. 55-80, 1948.

12. Mygind, S. H.: Ein Versuch zur Erklärung des sogenannten Regressions Phänomens (Recruitment). *Ztschr. f. Laryngol., Rhinol. and Otol.*, 29:277-285, June, 1950.

13. Watson, L. A.: A Manual for Advanced Audiometry. Minneapolis, Minn.: Colorcraft Press, 1949, pp. 30-33.

OBSERVATIONS ON THE LOCAL USE OF CORTISONE IN THE NOSE IN ALLERGIC RHINITIS.*

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As otolaryngologists we have been intrigued by the multiplicity of clinical conditions which have been improved by the parenteral use of cortisone and ACTH. Reports in the literature on allergic conditions, such as asthma, being improved, and polypi in the nose and sinuses disappearing after administration of ACTH and cortisone made us feel that one or both of these might prove of benefit in allergic rhinitis. Bordley[1] found that ACTH given parenterally has a decided effect on nasal mucosa and states "in those patients with pale edematous polypoid nasal membranes the edema rapidly disappeared and the color became bluish-pink." The exact mode of action of cortisone on the tissues is not definitely known, but it is believed that, as a result of a blocking or neutralizing effect on the antigen-antibody reaction, there might be a local tissue desensitization.

The division of ophthalmology of our hospital discovered that topical cortisone used locally in the eye was as successful as parenteral cortisone without side effects or the need for hospitalization. Steffensen[2] reports the use of cortone acetate‡ (1 cc. cortone acetate to 4 cc. of normal saline) gave no deleterious effect nor irritative eye symptoms, and no corneal or conjunctival atrophy was produced.

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‡The cortisone used was the 11-dehydro-17 hydroxycorticosterone acetate of Merck & Co., Rahway, N. J. We acknowledge with gratitude the cooperation of Dr. J. M. Carlisle, Medical Director of the Merck Laboratories.

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In view of this we were confident that a similar saline suspension of the cortone acetate used locally in the nose would have no deleterious effect on the nasal mucosa and might relieve allergic nasal symptoms, and the results of the ophthalmological treatment suggested there would be no systemic effect.

Since in its current usage the term "allergic rhinitis" is rather loosely descriptive of a congested or stuffy nose due to a variety of causes, we were critical in our choice of cases, selecting 25 only after careful scrutiny of a detailed past history, and after a complete ear, nose and throat examination and nasal smears were made. Only two of this group were children; 17 were men, six women. Of these cases, all had had nasal symptoms for many years; in many the diagnosis of allergic rhinitis had been made, either by other physicians or by us; several had asthma; 16 had nasal polypi, others had had polypectomies previously; 17 had been found sensitive on allergic studies; and most had had allergic treatment in one form or another, with or without benefit.

The experimental work began in April, 1950, and while the duration of therapy varied in each case, all patients were followed for several months. In order to observe any effect the warmer weather, with its tendency toward lessened infection, might have in the relief of nasal symptoms, the cortisone therapy in the majority of cases was discontinued during July and August.

In the preliminary phases of the investigation each patient underwent a four-hour period of observation in the clinic, during which time the nose was sprayed every half hour and detailed examination followed each application. No general reactions were noted. All patients experienced relief of nasal stuffiness and postnasal drainage. They were re-examined after a 24-hour interval and all reported they were free of any irritation and had had no local or general symptoms. The first few patients so treated were then given a quantity of the solution with instructions to spray each side of the nose four times a day at home and return for further clinical observation. No ill effects were noted and each of the patients

following in the series likewise used it at home. At each return visit the nose, sinuses, nasopharynx and pharynx were carefully examined. Nasal smears were routinely made.

Of the 25 treated, 12 (48 per cent) showed marked improvement; seven of these had had previous allergic studies with recurrence of symptoms, either because the specific therapy failed to produce relief or they had discontinued their allergic treatments; six (24 per cent) had some improvement, but were classified as doubtful; the balance (28 per cent) had no improvement and were considered as failures.

As we were largely dependent upon the subjective response to evaluate our therapy, our tabulation of the results may be open to criticism; however, we feel these results are not much different from those produced by specific allergy.

The following six cases we feel are representative of the series:

IMPROVED.

Case 1: E. J. Age nine, female, white. Case No. 505491. In 1947, a history was given of many colds, recurrent, blocked-up ears with a mild conductive deafness. A T. & A. in 1948 gave relief of the blocked-up ears with return to normal hearing. In 1948 and 1949 there were recurrent colds and stuffy nose.

In April, 1950, she again complained of a stuffy nose, blocked-up ears and cough. Examination revealed congested nasal mucosa which shrank readily with a decongestant, profuse mucus discharge in both nostrils and edema of the Eustachian orifices.

Nasal smear, April 11, 1950, showed 17 per cent eosinophiles; April 12, 1950, there were eosinophiles 14 per cent. Specific therapy was given in the form of the cortisone spray every half hour for three hours. April 13, 1950, she felt improved, and had less nasal congestion. Examination revealed only slight mucus discharge, and no edema of the Eustachian orifices. No eosinophiles were found on nasal smear. The following day the child awoke with slight deafness and nasal stuffiness, both of which cleared during the day with the use of cortisone nasal spray. April 15, 1950, there was a minimal amount of mucus discharge in the nasopharynx, no edema of Eustachian orifices, no coughing or sneezing. Nasal smear showed no eosinophiles. April 22, 1950, the child was symptom-free. Examination revealed a clean nose and clear sinuses. On awakening in the morning there was no phlegm in the throat. No further therapy was given.

On May 5, 1950, she was still symptom-free, without cough or nasal stuffiness. May 25, 1950, she was still symptom-free and her father, a physician, thought the "results were marvelous." Slight nasal stuffiness

and sneezing for two days were reported on June 8, 1950, but this cleared without therapy. June 23, 1950, there was again a slight nasal stuffiness which lasted two days. There were no accompanying ear symptoms. One nasal spray each day gave complete relief. The nasal smear showed 5 per cent eosinophiles. On nasal examination no noticeable congestion, discharge or atrophy were found.

Comment: This young girl with recurrent bouts of nasal stuffiness and clear nasal discharge, with elevation of eosinophile count, cleared with local cortisone spray. The father believes child has shown marked improvement. The recurring allergic nasal stuffiness might have cleared spontaneously, but one is impressed with the rapid disappearance of the nasal discharge and the absence of eosinophiles in the nasal smear following cortisone.

Case 2: Dr. M. J. Age 44, male, white. Case No. 412060. This patient had a history of repeated, frequent head colds, nasal congestion, nasal and postnasal discharge and chronic cough of many years' duration. Had considerable therapy and sinus irrigations on several occasions.

In 1944, the antra were dark on transillumination; mucus discharge was present in both nostrils and there was nasal congestion. X-rays disclosed thickening of mucous membranes of ethmoids and antra with a polyp in the left antrum.

Allergic studies showed four plus reaction to molds, house dust and bacteria; three plus to ragweed, timothy, orchard grass, June grass and red top.

Specific allergic therapy, hapamine injections, penicillin parenterally in courses gave no relief; slight relief was obtained with pyribenzamine. Smoking and occasional drinking were discontinued without change in symptomatology.

In May, 1950, he complained of continuous nasal congestion and marked postnasal discharge, requiring at least four handkerchiefs each day. Nasal mucosa was red and congested on examination but shrank readily with a decongestant. There was a profuse, clear mucoid discharge in both nostrils and nasopharynx. Nasal smear showed many polys and only a scattered eosinophile.

The cortisone nasal spray was begun, and he continued its use at home four times daily. One week later he reported a 50 per cent improvement, characterized by less nasal congestion and nasal discharge, requiring two instead of four handkerchiefs. The coughing was not only less frequent but also less severe.

In June the patient reported the good results persisted although the cough and symptoms recurred if the spray was discontinued. He was able to sleep through the night without coughing and thought his sense of smell might be returning.

He still felt improved in July. His cough was slight. He still required two handkerchiefs. Examination revealed a red, congested nasal mucosa, not greatly changed from the previous examination except for a somewhat lesser amount of discharge. The nasal smear showed 1 per cent

eosinophiles. Patient stated his symptoms continued to recur when the solution was discontinued, and he noted that the longer the solution was used, the longer the interval of relief when the spray was discontinued.

Comment: This physician with an allergic nose had had no benefit from any previous therapy. The symptoms were definitely relieved by cortisone, only to recur if the therapy was discontinued. The question is raised as to how long the therapy can be safely continued and whether or not the relief will be maintained.

Case 3: F. P. Age 58, male, white. Case No. 404353. In 1944, this man gave a history of sneezing, nasal and postnasal discharge and asthma. Examination at that time revealed a nasal polyp and chronic infection in the right antrum. Allergy studies revealed a four plus reaction to duck feathers, goose feathers, rabbit hair, giant ragweed, timothy and orchard grass; three to four plus delayed reaction to house dust, alternaria and to the bacterial proteins. Specific allergic therapy with improvement was given from 1944 through 1950.

He was seen on May 2, 1950, in our clinic, complaining of a stuffiness of the left ear. Examination showed a fairly marked nasal congestion, with numerous polypi in both nostrils, particularly on the right. The right eardrum was dull, retracted, but with no inflammatory reaction. Nasal smear showed many polymorphonuclear leucocytes but no eosinophiles.

The local cortisone therapy was begun on May 2, 1950. He returned May 23, 1950, at which time the polypi had almost completely disappeared. The patient stated he was at that time more comfortable than he had been for two years. The nasal cortisone spray was discontinued. On July 8, 1950, he returned for treatment because of a recurrence of symptoms. The nasal polypi were found to have returned to their original size. The cortisone nasal spray was resumed. By Aug. 28, 1950, the polypi had disappeared entirely. On Oct. 3, 1950, examination revealed a clean nose, no polypi, and clear sinuses. No evidence of atrophy of the nasal mucosa could be detected. The patient stated he had been using the cortisone nasal spray once daily; he was very markedly relieved of his nasal symptoms; and he was generally enthusiastic about the treatment.

Comment: One is impressed with the fact that the nasal polypi disappeared and with the rapidity of their disappearance upon the use of the cortisone spray as well as by the fact that the polypi recurred when the spray was discontinued.

DOUBTFUL IMPROVEMENT.

Case 4: H. A. Age 46, male, white. Case No. 247933. The history in this case dates back to childhood, with recurrent attacks of sneezing, nasal obstruction and nasal discharge. Three years previously he had had nasal polypi removed and a submucous resection and antromeatal windows done. Following this there was complete relief of nasal symptoms until one month prior to admission to our clinic.

When seen by us he complained of a head cold with marked right-sided nasal obstruction. Examination revealed the antromeatal windows to be patent. There were several small polypi bilaterally and one large polyp in the right nostril.

On May 23, 1950, cortisone nasal spray was begun, and continued daily. June 16, 1950, he stated he was breathing much more comfortably. On examination the polypi were found to be markedly decreasing in size. The therapy was continued. On June 30, 1950, he returned with a recurrence of the nasal symptoms and the polypi had returned to their original size. July 10, 1950, the polypi were removed under local anesthesia. The pathological diagnosis was "allergic nasal polypi." The patient failed to return for additional follow-up.

Comment: Although the patient was temporarily improved by the use of cortisone spray and his polypi showed some early tendency to decrease in size, the improvement was not maintained and the polyps returned to their original size even though the cortisone spray was continued.

Case 5: A. C. Age 37, male, white. Case No. 600298. This patient stated that since moving to Detroit, nine years previously, he had had sneezing, nasal stuffiness and nasal discharge. The nasal polypi had been removed two years and two and one-half years prior to this.

On March 4, 1950, he was treated by us for acute maxillary sinusitis and asthma, both of which responded to antibiotics and to sinus therapy which included sinus irrigations. April 17, 1950, examination revealed no evidence of sinus infection. There were several small polypi in both middle meati and a slight mucus discharge in both nostrils and in the nasopharynx. The patient was free of asthma but stated he had a tight feeling in his chest, more marked in the early morning. Nasal smear showed no eosinophiles. Allergic studies showed a three plus reaction to molds and bacteria.

On April 17, 1950, the cortisone nasal spray was begun, and he was treated every half hour until six treatments had been given. There was slight relief of his nasal symptoms and the patient thought his sense of smell had improved. April 21, 1950, he stated he had noted no change in his sense of smell, but the postnasal drainage was less and the tightness of his chest had been entirely relieved. April 28, 1950, he was symptom-free but again had had that morning a tight feeling in his chest. May 5, 1950, there was slight nasal stuffiness. The nasal smear was negative for eosinophiles.

He continued his therapy for six weeks with one to three-week intervals when no cortisone spray was used. He noted the spray gave him relief of the nasal symptoms, but that these recurred when the spray was discontinued. An "acute cold" contracted during this period was relieved in an hour by pyribenzamine 50 mgm. — the first time relief had been obtained with this drug.

Comment: This patient was given a weekly vaccine injection during this period, which he felt to be of doubtful benefit. His recurring nasal symptoms seemed to improve with cortisone, but we were unable to evaluate accurately the results of the therapy.

NO BENEFIT.

Case 6: C. W. Age 40, male, white. Case No. 386366. In this case nasal polypi had been removed several times in the past. In 1946, allergy studies showed a four and three plus reaction to corn, house dust, alternaria and bacteria. He was given specific allergic therapy and also hapamine injections with subjective improvement.

On Sept. 6, 1950, he came to our clinic with symptoms of sneezing, nasal obstruction and profuse rhinorrhea. Examination revealed a nose filled with polypi. The sinuses were clear on transillumination. The nasal smear was negative for eosinophiles.

He was given cortisone to be used four times a day. After a two-week trial without benefit he was unwilling to carry on with it any longer. Examination revealed no changes in the size of the polypi and at the patient's request they were removed. No further treatment with cortisone was given.

Comment: The period of treatment may not have been adequate, although in other patients the symptoms were usually relieved in this length of time.

DISCUSSION.

Cortisone, in a 1:4 dilution, appears to produce a more or less temporary local effect, sustained in proportion to the length of treatment, and which may be as beneficial perhaps as allergic therapy. Objectively, the use of the solution lessened the nasal secretions and decreased the edema of the nasal mucosa, even in those patients who felt they had experienced little or no relief. Surprisingly enough, many patients seemed to experience relief of mild asthmatic symptoms, such as wheezing, cough and tightness of the chest, though severe asthma remained unaltered. Headaches of allergic origin may be relieved. Polypi may be shrunk, or may disappear; in the latter it is too early to determine whether or not they will recur and, if so, what the interval before recurrence will be. There would appear to be no correlation between the presence or absence of eosinophiles in the nasal smear and the favorable or unfavorable response to the cortisone.

Comment: It is our present impression that the use of cortisone nasal spray produces a temporary relief of the nasal symptoms of allergic rhinitis. To date, no untoward general or local effects have been noted. No nasal mucosal atrophy can be discerned.

We are continuing our experiment in the hope of gathering further data on the effects of this drug when used locally in the nose. It may prove to be an adjunct in the diagnosis of allergic rhinitis. We hope to learn whether or not the effects obtained are permanent, particularly where the eradication of nasal polypi is concerned.

REFERENCES.

1. Bordley, J. E.; Harvey, A. McG.; Howard, J. E., and Newman, E. V.: Preliminary Report on the Use of ACTH in the Hypersensitivity State. Prac. First Clnc. ACTH Conference. Philadelphia, Pa.: Blakiston, 1950.

2. Steffensen, E. H.; Olson, J. A.; Margulis, R. R.; Smith, R. W., and Whitney, E. L.: The Experimental Use of Cortisone in Inflammatory Eye Disease. *Amer. Jour. Ophth.*, 33:1033-1040, July, 1950.

STENOSING PEPTIC ESOPHAGITIS. WITH A REPORT OF THE RESULTS OF TREATMENT IN 22 CASES.*†

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Esophageal stricture due to nonspecific esophagitis is a relatively rare but important cause of esophageal obstruction. During the past 20 years, 22 cases of stenosis due to esophagitis were admitted to the Stanford University, San Francisco County and St. Joseph's Hospitals in San Francisco. A study of the records shows that many of these patients were difficult problems in diagnosis and that treatment was often unsatisfactory.

Esophagitis in its mild form is the commonest disease of the esophagus and symptoms may consist of no more than heartburn or sour eructations;[42] however, as a complication of esophagitis there may occur a cicatricial stricture or a "peptic ulcer" of the esophagus, or both. It is important to avoid classifying these complications as separate disease entities. The cases to be reported here all developed esophageal stenosis as a result of esophagitis. They are being considered together as a group primarily because they provided the esophagologist and the surgeon with a specific type of problem in the diagnosis and treatment of esophageal obstruction. Before presenting this series of cases in detail, the etiology of esophagitis will be discussed and the differential diagnosis and treatment of strictures secondary to esophagitis will be reviewed.

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ETIOLOGY.

The pathological process under examination here is characterized by a nonspecific inflammatory reaction which begins in the esophageal mucosa and may extend to involve all layers of the esophageal wall. Single or multiple ulcers sometimes occur and can penetrate or even perforate the muscular coats. As in our cases, esophagitis may lead to intramural fibrosis and stricture. Esophagitis and the strictures resulting from it are practically always found at the distal end of the esophagus just proximal to the cardioesophageal junction.

It is now generally accepted that esophagitis is the result of the corrosive action of acid gastric juice on the esophageal mucosa.[3,10,33,45] Ferguson and his associates[22] have recently studied the effect of acid gastric juice on the esophageal, gastric and intestinal mucosa of animals and man. They found that the esophageal mucosa is especially vulnerable to damage by acid-peptic digestion and that gastric and intestinal mucosa are considerably more resistant.

Allison[3] also makes the point that the mucous membrane of the esophagus has a low natural resistance to peptic digestion. He then calls attention to the important rôle of the mechanism at the cardia of the stomach which normally prevents gastric juice from flowing back into the esophagus — except to a limited degree.[35] Conditions which decrease or abolish the valve-like action at the gastric cardia will favor an increased reflux of stomach secretions into the esophagus. In some such cases, acid gastric juice may be expected to cause inflammation of the esophagus.

Actually, it does appear that esophagitis is almost always associated with circumstances which in one way or another reduce the effectiveness of the mechanism of the gastric cardia. In this category will be found hiatal herniae, in-lying gastric tubes, prolonged vomiting, comitose or moribund states. All of these are known to predispose to esophagitis.

The frequent coincidence of esophagitis and hiatal hernia of the short esophagus type deserves special comment. Short esophagus with a sliding hiatal hernia may be either congeni-

tal or acquired. In most instances, the abnormality is probably acquired.[3,23,36] Dey *et al.*[18] made some pertinent and interesting observations in dogs. By traction upon the liver or gall-bladder, by stretching the wall of the gall-bladder, cystic duct or stomach, or by stimulating the vagus nerve, they were able to cause such reflex shortening of the esophagus that the cardia of the stomach was pulled up into the mediastinum. A short esophagus with hiatal hernia was thus temporarily produced. It has been suggested that an irritant focus below the diaphragm in man (for example, chronic cholecystitis,[21] peptic ulcer or gastric cancer) may cause similar reflex shortening of the esophagus with hiatal hernia. Such an hiatal hernia would reduce the effectiveness of the mechanism of the cardia and permit reflux of gastric juice into the lower esophagus as a prelude to the development of esophagitis. Smithers[36] believes that in most cases seen by the radiologist, the shortened esophagus and hiatal hernia are due to contraction of the esophageal musculature and that normal relationships can be re-established by the relaxation of esophageal muscle spasm. In some instances, however, there may occur sufficient fibrosis from esophagitis to make permanent the esophageal shortening and hiatal hernia. Further progression of the esophagitis can lead to ulcer or stricture of the esophagus, or both.

Many patients with esophagitis have a peptic ulcer in the duodenum or stomach or give a history consistent with previous ulcer. In view of present concepts of the etiology of esophagitis, it is not surprising to learn that the peptic ulcer diathesis with its attendant hyperacidity, hypersecretion and vagotonia is a not uncommon background for esophagitis. Wangensteen and Leven[44] think that esophagitis is similar to duodenal ulcer in origin. They suggest that, next to duodenal ulcer, esophagitis should be considered the commonest manifestation of ulcer disease.

The triad of esophagitis, duodenal ulcer and hiatal hernia of the short esophagus type occurs clinically with striking frequency.[9] The development of these three conditions in the

same patient must be more than a coincidence and serves as an example of the inter-relationships discussed in the foregoing paragraphs.

It has been noted that significant numbers of patients with strictures from esophagitis are alcoholics[9] (see Table 2). It is likely that a high alcoholic intake in some way results in an increased tendency to develop esophagitis.

For our purposes we will accept the following propositions:

1. Acid gastric juice is the irritant responsible for esophagitis.
2. The esophageal mucosa is unusually susceptible to acid-peptic digestion.
3. When the mechanism of the gastric cardia becomes ineffective, reflux of gastric juice into the lower esophagus increases and esophagitis may result.
4. Treatment should logically be directed toward reducing gastric acidity or keeping gastric juice out of the esophagus, or both.

DIFFERENTIAL DIAGNOSIS.

As stated above, esophagitis is not uncommon and generally causes minimal discomfort. The complications of esophagitis, however, produce more definite symptoms. When ulceration of the esophagus (usually described as "peptic ulcer of the esophagus") occurs, moderate epigastric or substernal pain and possibly some dysphagia due to esophageal spasm may be the chief complaints. Massive hemorrhage or chronic blood loss can take place and, rarely, the ulcer will perforate the esophagus with devastating results.

When esophagitis leads to stricture, the outstanding symptom is dysphagia. The development of swallowing difficulties is at times preceded for a variable period by heartburn, substernal distress or symptoms suggestive of peptic ulcer. The patient may indeed have a co-existing peptic ulcer in duodenum, stomach or esophagus. On the other hand, painless and progressive dysphagia can be the only complaint.

Stenosing esophagitis may occur at any age. It is very rare in infants and children in whom it is secondary to a congenitally shortened esophagus with hiatal hernia.[3] These strictures are most frequently seen in the older age group, the majority of patients being men over 50. Inability to eat satisfactorily usually results in weight loss which may be considerable. Painful dysphagia and hematemesis or melena suggest that both stricture and ulceration are present. There are no characteristic physical findings.

As a general rule, the clinician seeing the patient with stenosing peptic esophagitis for the first time will also have to consider in his differential diagnosis various other causes of distal esophageal obstruction. The most important of these are traumatic strictures (due most often to chemical burns), achalasia, and neoplasm of the esophagus or stomach.

The development of dysphagia following the ingestion of lye or other chemicals plus the radiological and endoscopic evidences of a benign esophageal stricture are usually enough to make the diagnosis obvious. There is little likelihood that strictures of this type will be confused with those due to esophagitis.

Achalasia of the esophagus (also known as idiopathic dilatation, megaesophagus and cardiospasm) is seen predominantly in women, occurs also at all ages and accounts for about 20 per cent of cases of dysphagia.[28] It is important to point out that achalasia and stenosing peptic esophagitis are not the same condition nor do they represent different stages of the same disease. In achalasia there is dilatation, hypertrophy and lengthening of the esophagus without organic obstruction. The distal 3 to 6 cm. of the esophagus are relatively narrowed and classically there is no fibrous stricture. It is true that inflammation may result from retained food. Also, it is of special interest that peptic esophagitis frequently develops in patients with achalasia after cardioplastic operations which make possible the reflux of gastric juice into the esophagus.[6,32] In stenosing esophagitis, the esophagus shows minimal or no dilatation and, instead of being lengthened, the esophagus is often shortened with an associated hiatal hernia.

TABLE 1.

OPERATIONS FOR STENOSING PEPTIC ESOPHAGITIS (EXCLUSIVE OF GASTROSTOMY) AS REPORTED IN THE MEDICAL LITERATURE.

Author	Operation	No. of Cases	Result	Comment
Eggers, 1935[19]	Resection, esophagus with construction, antethoracic esophagus	1	Good	Followed 16 years[20]
Clark and Adams, 1945[13]	Esophagogastrostomy	3	Good	Followed a few weeks to a few months
Allison, 1948[3]	Resection, esophagus with esophagojejunostomy	5	Good, 4	Duration follow-up not stated. One postoperative death.
Mason, 1948[29]	Plastic on esophageal stricture (Heineke-Mikulicz)	1	Good	Followed six months
Sweet, 1949[38]	Plastic on esophageal stricture (Heineke-Mikulicz)	6	Good, 4 Fair, 2	Duration of follow-up not stated
Wangensteen and Leven, 1949[44]	Partial gastrectomy	7	Good	Followed 10 months to 8½ years[43]
Mautz, 1946[30]	Resection, esophagus with Esophagogastrostomy	1	Good	Followed 3 years
Wyndham, 1947[46]	Resection, esophagus with Esophagogastrostomy	1	Good	Followed 3 months
Haight, 1948[24]	Resection, esophagus with Esophagogastrostomy	1	Good	Followed 4 years[1]

Dailey, 1948[15]	Resection, esophagus with Esophagogastrostomy	3	Good, 2	Two patients followed 2½ years. One post-op. death[16]
Baumann, 1948[8]	Resection, esophagus with Esophagogastrostomy	1	?	No follow-up reported
Keeley, 1948[27]	Resection, esophagus with Esophagogastrostomy	1	Good	Followed 1½ years
De Amesti and Chechilnitzky, 1948[17]	Resection, esophagus with Esophagogastrostomy	1	Fair	Followed 7 months
Sweet, 1949[38]	Resection, esophagus with Esophagogastrostomy	20	Good, 19	Duration of follow-up not stated. One post-op. death
Case Records, MGH, 1949[11]	Resection, esophagus with Esophagogastrostomy	1	——	Post-operative death
Stephens, 1949[37]	Resection, esophagus with Esophagogastrostomy	1	Fair	Duration of follow-up not stated
Van Aken, 1950[40]	Resection, esophagus with Esophagogastrostomy	2	Good	Duration of follow-up not stated
Alves, 1950[5]	Resection, esophagus with Esophagogastrostomy	1	Good	Duration of follow-up not stated

Total number of resections with esophagogastrostomy for stenosing peptic esophagitis—34.
Postoperative deaths—3.
Operative mortality—9%.

Results of treatment in 31 survivors of resection: Good in 28, or 90%.
Fair in 2, or 6%.
Result not stated in 1, or 4%.

TABLE 2.

FINDINGS ASSOCIATED WITH STENOSING PEPTIC ESOPHAGITIS IN 22 PATIENTS.

	Number of Patients	Per Cent
Peptic Ulcer in the Stricture	9	41
Esophagitis Without Demonstrable Ulcer	13	59
Hiatal Hernia (All Types)	13	59
Hiatal Hernia of Short Esophagus Type	9	41
Evidence of Peptic Ulcer in Duodenum (9) or Stomach (1)	10	45
Combination of: Esophageal Stricture, Hiatal Hernia of Short Esophagus Type, and Evidence of Duodenal or Gastric Ulcer	6	27
History of Heavy Alcohol Intake	7	32

TABLE 3.

RESULTS OF CONSERVATIVE TREATMENT IN 11 PATIENTS WITH STENOSING PEPTIC ESOPHAGITIS.

Result	No. of Cases	Comment
Good	3	Follow-up periods of one and one-half, four and seven years.
Poor	6	*Two* patients, followed two and seven years, require treatment intermittently and are incapacitated (both are alcoholic).
		Three patients continued to have symptoms until they died of intercurrent diseases in two, nine and 10 months. It is probable that the malnutrition resulting from their esophageal strictures was a contributing factor in their early deaths.
		One patient died of mediastinitis after an esophagoscopy.
Unknown	2	Asymptomatic a few weeks after initial treatment, but no later follow-up information obtainable.

TABLE 4.
RESULTS OF SURGICAL TREATMENT IN 11 PATIENTS WITH STENOSING PEPTIC ESOPHAGITIS.

Indications for Operation	Name	Age	Sex	Operation	Result	Comment
Failure of conservative measures: 5 cases	J.J.	62	M	Gastrostomy	Good	Followed 1½ years
	E. L.	57	M	Gastrostomy	Poor	Died of hemorrhage from esophageal ulcer 10 years after operation
	E. C.	68	M	Gastrostomy	Poor	Postoperative death
	P. N.	69	M	Transthoracic repair of hiatal hernia, short esophagus type	Poor	Steadily declined, dysphagia continued, died 13 months after operation
	J. M.	67	M	Gastrostomy, then plastic to esophageal stricture (Heineke-Mikulicz), then left phrenic nerve interruption	Poor	Followed 6 years. Has persistent dysphagia, pain and repeated esophageal hemorrhages
Bleeding duodenal ulcer: 2 cases	H. K.	49	M	Partial gastrectomy	Good	Required one bouginage postoperatively but is now 21 months since operation and eating normally
	A.K.	77	M	Partial gastrectomy	Poor	Postoperative death
Preoperative diagnosis of carcinoma of esophagus: 4 cases	F. H.	51	M	Transthoracic exploration of the esophagus with bouginage under direct vision	Poor	Failed steadily, dysphagia continued, died 9 months after operation
	J. M.	59	M	Transthoracic exploration of the esophagus with "excision of periesophageal scar tissue"	Poor	Followed 4 years. Still requires bouginage and has occasional esophageal hemorrhage
	H. S.	51	M	Plastic to esophageal stricture (Heineke-Mikulicz) and vagotomy	Poor	Followed 1 year. Recurrence of dysphagia and heartburn
	F. L.	36	M	Resection esophagus and esophagogastrostomy	Good	Symptom-free for 10 months. Patient died with acute alcoholism. Autopsy showed no evidence of recurrent esophagitis

The most difficult and important problem is to distinguish stenosing peptic esophagitis from carcinoma of the distal esophagus or gastric cardia. There is abundant evidence in the literature[8,11,17,19,27,31] and in the cases to be reported here (see Table 4) that this has sometimes proven difficult. When a history of dysphagia over a period of years is obtained, this, of course, favors a benign stricture; however, the symptoms of stenosing esophagitis and obstructing cancer may be quite the same and consist merely of dysphagia for a few weeks or months plus weight loss. At the San Francisco County Hospital, cancer was the cause of dysphagia almost 15 times as often as esophagitis. The relative infrequency of stenosing esophagitis as compared with malignant obstruction was probably responsible in our series for a failure to consider seriously the diagnosis of esophagitis preoperatively in cases which, in retrospect, were quite typical.

Roentgenographic examination in stenosing peptic esophagitis will disclose a partial or complete obstruction at the distal end of the esophagus. Specifically, the esophagram usually shows:

1. A proximal esophagus of normal size or with only minimal dilatation;
2. A funnel-shaped narrowing of the distal esophagus (without shelving margins) leading into —
3. An area of organic stricture of variable length (usually several centimeters) in which there is distortion or destruction of the mucosal pattern and sometimes definite ulceration.

The esophagus will frequently be shortened and associated with a hiatal hernia of the sliding type so that the cardioesophageal junction lies in the mediastinum, occasionally as high as the root of the lung (see Figs. 1-5). Hiatal hernia of another type may be found in some cases and examination of the stomach and duodenum may show signs of active or healed peptic ulceration. The degree of esophageal obstruction may be increased by an element of esophageal spasm.

Carcinoma of the distal esophagus or gastric cardia with infiltration of the esophageal wall usually produces a Roentgen picture of constriction which begins fairly abruptly, often with shelving margins and evidence of an irregular filling defect in the lumen of the esophagus. The esophagus above obstructing cancer may be dilated but usually is not. It should

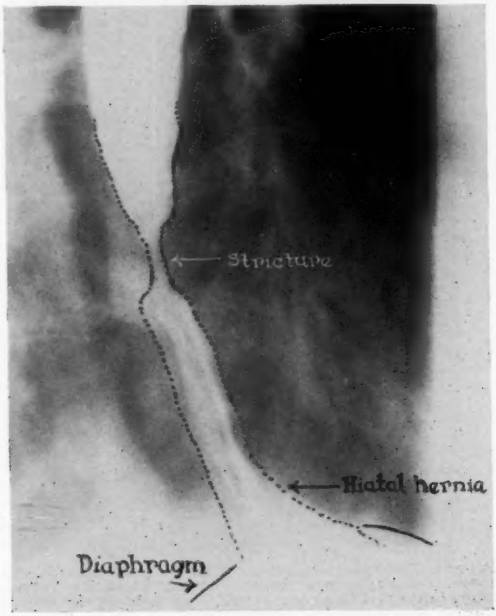

Fig. 1. Patient C. M., aged 67, male. Diagnosis: Stenosing peptic esophagitis. History: Dysphagia, intermittent, eight years. Chronic alcoholic. Treatment: Diet and repeated bouginage. Result: Poor. Followed two years.

be kept clearly in mind that it is impossible for the Roentgenologist to rule out cancer with absolute certainty even though the esophagram may be typical of stenosing esophagitis. Not only has malignancy been known to develop in a stricture due presumably to esophagitis[9] but carcinoma is also occasionally associated with short esophagus and hiatal hernia.[36,39]

Esophagoscopy is an essential diagnostic procedure in the usual case of stenosing esophagitis. Not only is it important to establish the diagnosis as a basis for therapy but the consequencies of overlooking resectable cancer are disastrous to the patient. While the gross appearance of the lesion at endoscopy may be indistinguishable from carcinoma, the changes

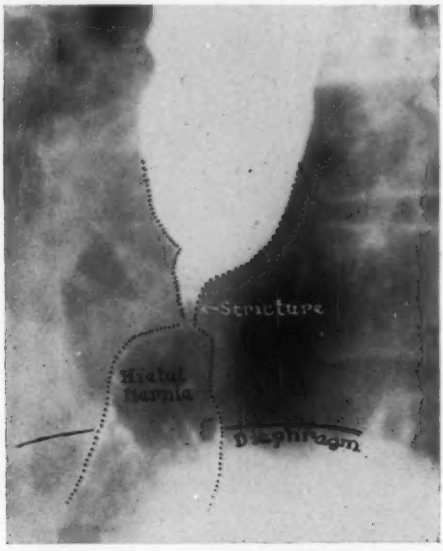

Fig. 2. Patient F. H., aged 51, male. Diagnosis: Stenosing peptic esophagitis. History: Dysphagia, one week. Chronic alcoholic. Treatment: Exploratory thoracotomy with preoperative diagnosis of carcinoma. Since no neoplasm found at surgery, esophagus not removed. Result: Died nine months postoperatively with continued severe dysphagia.

seen in the esophagus are frequently quite typical. In obstruction due to esophagitis the mucous membrane often shows acute congestion for several centimeters above the stenosis, often with superficial discrete ulcers or extensive denudations. These acute superficial changes above the obstruction are important for they usually indicate a benign rather than a malignant lesion. Granulation tissue may be seen at the stricture which feels softer and more uniform

than malignancy as a bougie is passed through it. When the Roentgenograms have been suggestive of cancer, the irregularities seen through the esophagoscope are found to be produced by extensive ulceration with granulation tissue.[3]

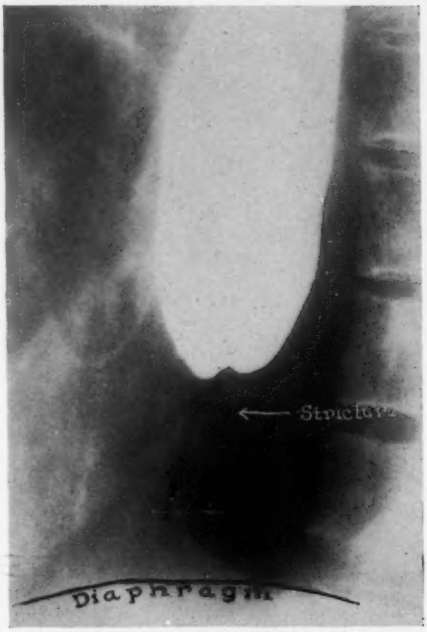

Fig. 3. Patient J. M., aged 67, male. Diagnosis: Stenosing peptic esophagitis. History: Dysphagia and hematemesis, three months. Treatment: Gastrostomy followed by transthoracic plastic to stricture (Heineke-Mikulicz), then left phrenic nerve interruption. Hiatal hernia and short esophagus found at operation. Result: Poor. Has persistent dysphagia and repeated esophageal hemorrhages. Followed six years.

Stephens[37] points out that the endoscopist can make the diagnosis of short esophagus and hiatal hernia when these are present by identifying the cardioesophageal junction well above the diaphragm. Biopsies immediately above the stricture will show esophageal mucosa, while those just below will contain gastric mucosa. In the last analysis, it is only by

obtaining multiple negative biopsies that carcinoma can be ruled out with reasonable certainty. It has been emphasized that some biopsies should, if possible, be taken from deep within the stricture[9] and repeated endoscopies may be necessary. Finally, when a suspicion of cancer remains, an exploratory operation should be done.

Fig. 4. Patient H. S., aged 51, male. (Private patient of Dr. Nelson J. Howard.) Diagnosis: Stenosing peptic esophagitis. History: Dysphagia, four months. Treatment: Carcinoma of the esophagus suspected preoperatively. Transthoracic plastic to stricture (Heineke-Mikulicz) plus vagotomy. Result: Poor. Heartburn and some dysphagia persist. Followed one year.

TREATMENT.

Medical measures of value in esophagitis are bland, soft diet, frequent feedings, antispasmodics, antacids and mild sedation as for peptic ulcer. Elevation of the head of the bed to reduce the reflux of gastric juice into the esophagus while sleeping is advisable.

When a stricture prevents adequate food intake, endoscopic dilatation is required. It may be necessary to repeat this at varying intervals for many months as the stricture has a tendency to close down again gradually. Benedict and Sweet[9]

found that bouginage and dietary treatment gave satisfactory results in over two-thirds of their 60 cases of stricture due to esophagitis. Certainly a thorough trial of this regimen is indicated.

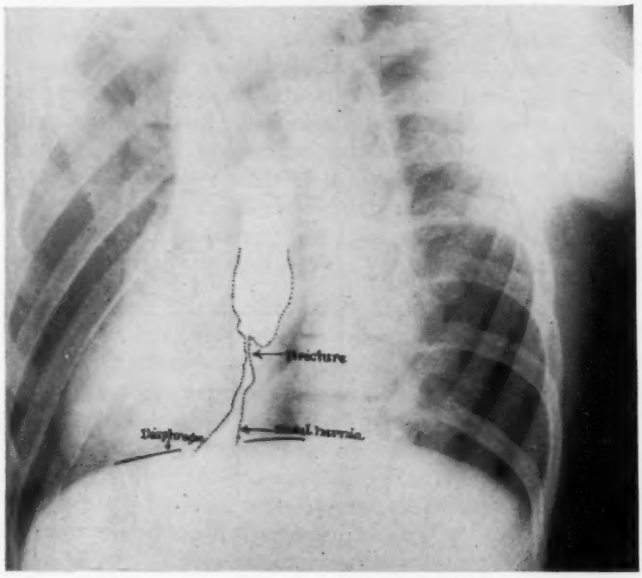

Fig. 5. Patient F. L., aged 36, male. (Referred to author by Dr. Wesley E. Scott.) Diagnosis: Stenosing peptic esophagitis. History: Progressive painless dysphagia, three months. Treatment: Preoperative diagnosis of esophageal carcinoma made. Benign stricture found at operation. Esophageal resection and esophagogastrostomy carried out. Result: Good, but patient died of acute alcoholism 10 months postoperative. Autopsy showed no evidence of esophagitis.

Surgical treatment should be considered when there is *1.* failure of repeated dilatations to relieve marked dysphagia, *2.* recurrent severe esophageal hemorrhage, *3.* intractable pain, or *4.* esophageal perforation. Many of the patients with stenosing esophagitis are elderly and effective surgical measures are not without risk. Since the indications for surgery are to a certain extent relative, each case should have careful individualized evaluation before an operation is under-

taken. There is no unanimity of opinion regarding the operation of choice for stricture due to esophagitis. A discussion of the various procedures which have been tried in this condition is, therefore, in order.

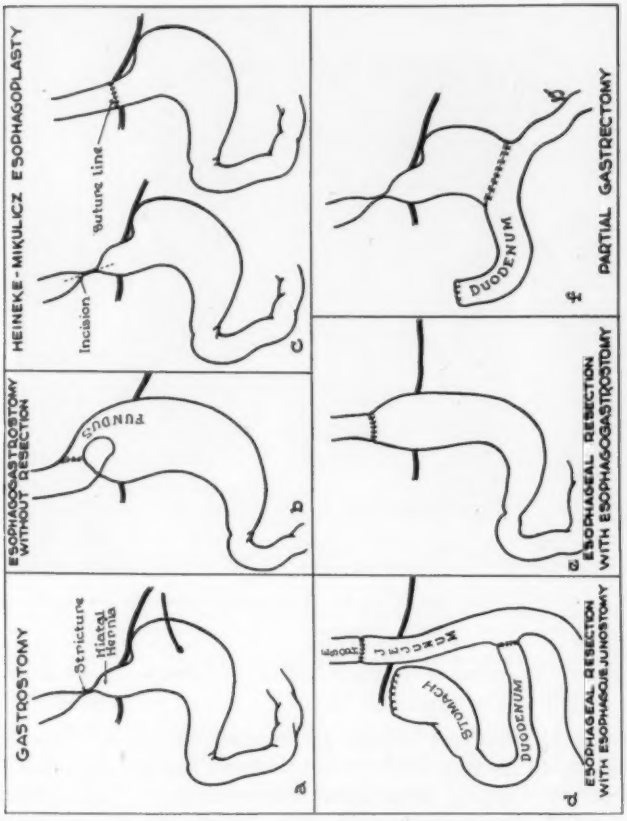

Fig. 6. Operative procedures which have been used for stenosing peptic esophagitis. See text for discussion.

Gastrostomy: The simplest operation which has been used is gastrostomy (see Fig. 6a). This permits restoring the patient to better nutritional state but will not necessarily have any influence on the stricture.[31,44] It should now rarely, if

ever, be necessary to resort to gastrostomy since parenteral alimentation is so effective, and the insertion of a small polyethylene tube into the stomach at endoscopy will permit adequate feeding for prolonged periods while conservative measures are being tried.

Esophagogastrostomy: Clark and Adams[13] have successfully employed an anastomosis of the fundus of the stomach to the esophagus above the level of the stricture (see Fig. 6b). This procedure will not prevent continued contact of acid gastric juice with the esophagus at the site of stricture and above it. It is conceivable that the inflammatory process in the esophagus might, therefore, extend upward to involve the region of the anastomosis. For this reason, the operation has theoretical disadvantages which do not recommend it for general use.

Esophagoplasty: Heineke-Mikulicz type of plastic procedure (see Fig. 6c) has been done by Sweet[38] in six patients who had short strictures. All patients were relieved of dysphagia, but two have complaints of heartburn and eructation of gastric contents. The result was very poor in the two cases in our series in which this operation was done (see Table 4). Both patients, one of whom had a vagotomy at the time of the plastic, developed heartburn postoperatively and dysphagia occurred. Since esophagoplasty neither reduces gastric acidity nor prevents reflux of gastric juice into the esophagus, it seems reasonable to predict that the long-term results will be unsatisfactory in a high percentage of cases treated in this manner.

Repair of Hiatal Hernia: It would seem logical to attempt to restore the competency of the mechanism of the cardia by repairing a hiatal hernia should one be present. When the stricture is not severe and the hernia is of the paraesophageal or sliding type *without* a short esophagus, this may be successful.[3] When there are extensive cicatricial changes and the esophagus is shortened by fibrosis, it is difficult to replace the esophagogastric junction below the diaphragm. Consequently, it is generally considered that hiatal herniae of the

short esophagus type are not easy to repair satisfactorily.[25,34] Since most herniae associated with stenosing esophagitis are of the short esophagus type, repair of hiatal hernia has a limited usefulness but may be indicated in properly selected cases.

Partial Gastrectomy: Wangensteen and Leven[44] have reported good results in the treatment of seven cases of stenosing peptic esophagitis by a resection of three-fourths of the stomach as for duodenal ulcer (see Fig. 6f). They stress the importance of reducing gastric acidity and decreasing gastric emptying time. Certainly in their patients the response to gastrectomy was gratifying and supported their thesis that esophagitis is of acid-peptic origin.

Castleton and Dolowitz[12] noted the prompt healing of a bleeding peptic ulcer of the esophagus after a gastric resection. One of our patients with stenosing esophagitis was relieved of dysphagia following a partial gastrectomy for duodenal ulcer (see Table 4). On the other hand, stricture due to esophagitis has been seen to occur *after* a gastric resection for ulcer. Two such cases have been observed by Allison[2,4] and one by Van Aken.[40] The details of these resections with estimates of the amount of stomach removed and the postoperative levels of gastric acidity are not reported. One is, therefore, not entitled to infer that gastric resection will not give satisfactory results if done according to Wangensteen's specifications; nevertheless, these cases do suggest that the place of partial gastrectomy in the treatment of stenosing esophagitis requires further evaluation based on the study of a greater number of patients.

Esophageal Resection and Esophagogastrostomy (see Fig. 6e): This operation has been reported more frequently than any other in the treatment of these benign strictures (see Table 1). Sweet[38] has by far the greatest recorded personal experience with the surgical management of this disease. In 20 patients he did an esophageal resection and esophagogastrostomy with one postoperative death (due to coronary occlusion), an operative mortality of 4 per cent. The remaining

patients have been relieved of dysphagia, although a few developed digestive discomforts. These are not described in detail nor is the duration of follow-up given. Sweet considers a wide resection of the stricture important in order to get above any inflammatory reaction in the esophageal wall which might dispose to recurrence of esophagitis and stricture.

Since good or fair results have been reported in 96 per cent of the 31 cases which survived resection and esophagogastrostomy (see Table 1), it would seem that this procedure is better established than any other as a satisfactory operation for stenosing peptic esophagitis.

Esophageal Resection and Esophagojejunostomy (see Fig. 6d): Allison[3] has considered the use of vagotomy in the treatment of peptic esophagitis but has rejected the procedure because he has seen peptic ulcer develop in the esophagus a few months after vagotomy for gastric ulcer; furthermore, vagotomy does nothing to alter the basic anatomical deformity which prevents proper function of the mechanism of the cardia and vagotomy will predispose to reflux by prolonging gastric emptying time. He has noted the development of ulcers in the esophagus in four of 21 patients following partial esophagogastrectomy with mediastinal anastomosis for carcinoma and these ulcers seemed to bear no relation to the acid secretion in what was left of the stomach. This has led him to stress the primary importance of the mechanism of the gastric cardia. He suggests that the best protection from a recurrence of esophagitis is afforded by resection and esophagojejunostomy rather than esophagogastrostomy. Allison has done a resection and esophagojejunostomy in five patients with one postoperative death. One died a year later of tuberculosis, but was apparently free of symptoms of esophagitis. The other three patients remain symptom-free, but the duration of follow-up was not given.

Esophagojejunostomy will indeed effectively prevent contact of gastric juice with esophageal mucosa and relief from esophagitis might be uniformly expected; however, a certain proportion of patients so treated is likely to develop serious

digestive complaints as a result of the loss of the gastric reservoir and the passage of food from esophagus directly into the small intestine. More experience with this interesting procedure will be necessary before its possible undesirable side effects can be determined.

REPORT OF CASES.

In the 22 cases to be reported here, the diagnosis of stenosing peptic esophagitis was made by Roentgenography plus esophagoscopy (usually with biopsy) or by operation or autopsy. The stricture was just proximal to the cardioesophageal junction in all cases. There were 18 males and four females. They ranged in age from 36 to 81 years and 18 (or 82 per cent) were over 50 years of age.

The chief complaint of all patients was dysphagia. Gastrointestinal hemorrhage which varied from one-half cupful of fresh blood to massive losses occurred in eight patients (or 36 per cent). One patient died of hemorrhage from a peptic ulcer in his esophageal stricture.

The findings associated with stenosing esophagitis are shown in Table 2. The high incidence of hiatal hernia, short esophagus, peptic ulcer disease, and alcoholism is of interest and the implications of this have already been commented upon. Studies of gastric acidity were so frequently impossible or impracticable because of the esophageal obstruction that one can draw no conclusions from these cases on the relation of acidity to esophagitis.

Eleven patients were treated conservatively (see Table 3) and 11 were operated upon (see Table 4). A result was considered "good" if the patient was eventually relieved of symptoms, "fair" if he was improved but not asymptomatic, and "poor" if he was no better, worse, or relapsed promptly after temporary improvement.

Discussion: This series of cases is small and the high incidence of poor results has no statistical significance as regards

the outlook for patients with stenosing esophagitis in general. It should be pointed out that all except two of these patients were from public clinic services. Many were first seen in a late stage of their disease with serious malnutrition and other complicating illnesses. Some had neither the resourcefulness nor the inclination to cope with their problem.

There were two other circumstances which influenced the results in the group of patients treated surgically. The first of these was the failure to make a correct preoperative diagnosis. In four of the 11 surgical cases, an incorrect diagnosis of carcinoma of the esophagus led to an exploratory operation which might have been deferred for a trial of conservative management. These cases were all sufficiently characteristic of stenosing peptic esophagitis to have stimulated further study had this condition been seriously considered. This limited experience emphasizes the fact that, although the clinical picture of stenosing esophagitis may closely resemble that of carcinoma, a careful differentiation is important in order to spare the patient unnecessary surgery.

Secondly, results in our surgically treated patients were influenced by an uncertainty on the part of the surgeons as to what constitutes an adequate operation for stenosing peptic esophagitis. This is not surprising since the condition is relatively rare and operative procedures are not standardized.

On the basis of the evidence so far reported, it seems reasonable to advise wide resection of the stricture and esophagogastrostomy as the most satisfactory operation for stenosing esophagitis. The vagotomy which accompanies this procedure plus the displacement of the fundus into the chest apparently combine to reduce gastric acidity and minimize reflux into the esophagus to such an extent that almost all patients so treated will remain symptom-free.

Finally, stenosing peptic esophagitis is a serious and sometimes fatal condition which is probably in many instances a manifestation of the peptic ulcer diathesis. Ulcer disease is notorious for its chronicity and tendency to remission and

exacerbation. The results of surgical treatment of strictures due to esophagitis must, therefore, be accepted with caution unless based on follow-up of five or more years. Since most reports in the literature give only the early results of surgery, conclusions regarding the operation of choice are necessarily tentative.

SUMMARY.

1. Esophagitis is a common disease of the esophagus which may lead to ulceration, stricture, or both.

2. Reflux of acid gastric juice into the distal esophagus is the probable cause of esophagitis. The esophageal mucosa is especially vulnerable to acid-peptic digestion.

3. Hiatal herniae and other conditions which reduce the effectiveness of the mechanism of the cardia predispose to reflux of gastric juice into the esophagus and, therefore, to esophagitis.

4. When peptic esophagitis results in esophageal stenosis, bouginage plus medical measures as for peptic ulcer will give relief in most cases.

5. When conservative measures fail, esophageal resection with esophagogastrostomy is the operative procedure of choice.

6. Twenty-two cases of stenosing peptic esophagitis are reported and results of treatment discussed.

NOTE: The author acknowledges his appreciation to Nelson J. Howard, M.D., and to the University of California, Department of Surgery, at the San Francisco County Hospital for permission to include their patients in this survey.

REFERENCES.

1. ALEXANDER, JOHN: Personal communication.

2. ALLISON, P. R.: Peptic Ulcer of the Esophagus. *Jour. Thoracic Surg.*, 15:308-317, Oct., 1946.

3. ALLISON, P. R.: Peptic Ulcer of the Esophagus. *Thorax*, 3:20-42, Mar., 1948.

4. ALLISON, P. R.: Personal communication.

5. Alves, J. de R.: Ulcera Péptica de Esôfago. *Brasil-Medico*, 64:61-65, Mar.-May, 1950.

6. Barrett, N. R., and Franklin, R. H.: Concerning the Unfavorable Late Results of Certain Operations Performed in the Treatment of Cardiospasm. *Brit. Jour. Surg.*, 37:194-202, Oct., 1949.

7. Bartels, E. C.: Acute Ulcerative Esophagitis. A Pathologic and Clinical Study of 82 Cases Observed at Necropsy. *Arch. Path.*, 20:369-378, Sept., 1935.

8. Baumann, J.: Retricissement de l'oesophage traite par resection. *Mem. d. L'Acad. d. Chir.*, 74:309, Apr., 1948.

9. Benedict, E. B., and Sweet, R. H.: Benign Stricture of the Esophagus with Special Reference to Esophagitis, Hiatus Hernia, Esophageal Ulcer and Duodenal Ulcer. *Gastroenterology*, 11:618-628, Nov., 1948.

10. Butt, H. R., and Vinson, P. P.: Esophagitis. I—Anatomy and Physiology and a Review of the Literature. *Arch. Otolaryngol.*, 23:391-413, Apr., 1936.

11. Case Records of Massachusetts General Hospital. Case 35031. *New Eng. Jour. Med.*, 240:107-111, Jan. 20, 1949.

12. Castleton, K. B., and Dolowitz, D. A.: Peptic Ulcer of the Esophagus with Severe Bleeding Controlled by Oxycel. *Gastroenterology*, 10:787-800, May, 1948.

13. Clark, D. E., and Adams, W. E.: Transthoracic Esophagogastrostomies for Benign Strictures of the Lower Esophagus. *Ann. Surg.*, 122: 942-953, Dec., 1945.

14. Cushing, H.: Peptic Ulcers and the Interbrain. *Surg., Gynec. and Obst.*, 55:1-34, July, 1932.

15. Dailey, J. E.: Discussion of article by J. W. J. Strieder. *Jour. Thoracic Surg.*, 17:173-174, Apr., 1948.

16. Dailey, J. E.: Personal communication.

17. De Amesti, F., and Chechilnitzky, L.: Ulcera Peptica Estenosante del Esofago, Tratada por Esofagos-gastrectomia. *Rev. Med. d. Chile*, 76: 232-235, Mar., 1948.

18. Dey, F. L.; Gilbert, N. C.; Trump, R., and Roskelley, R. C.: Reflex Shortening of the Esophagus in the Experimental Animal with the Production of Esophageal Hiatus Hernia. *Jour. Lab. and Clin. Med.*, 31:499-506, May, 1946.

19. Eggers, C.: Resection of Thoracic Portion of Esophagus for Chronic Ulcer. *Ann. Surg.*, 101:940-944, Mar., 1935.

20. Eggers, C.: Personal communication.

21. Feldman, M.: Esophageal Dysphagia, Associated with Gall-Bladder Disease. *Rev. Gastroenterology*, 17:1044-1047, Nov., 1950.

22. Ferguson, D. J.; Sanchez-Palomera, E.; Sako, Y.; Clatworthy, H. W., Jr.; Toon, R. W., and Wangensteen, O. H.: Studies on Experimental Esophagitis. *Surg.*, 28:1022-1039, Dec., 1950.

23. Fincher, E. F., and Swanson, H. S.: Esophageal Rupture Complicating Craniotomy Symptom Complex and Proposed Surgical Treatment. *Ann. Surg.*, 129:619-630, May, 1949.

24. Haight, C.: Discussion of article by A. M. Olsen and S. W. Harrington. Ref. No. 43.

25. Harrington, S. W.: Diagnosis and Treatment of Various Types of Diaphragmatic Hernia (Special Monograph). *Amer. Jour. Surg.*, 50:377-446, Nov., 1940.

26. Jackson, C.: Peptic Ulcer of the Esophagus. *Jour. A. M. A.*, 92:369-372, Feb. 2, 1929.

27. Keeley, C. D.: Benign Strictures of the Lower Esophagus Treated by Resection and Esophagogastric Anastomosis. *Canad. Med. Assn. Jour.*, 59:369-370, Oct., 1948.

28. Maingot, R.: Techniques in British Surgery. Philadelphia-London: W. B. Saunders Co., 1950, pp. 358-372.

29. Mason, J. L.: The Surgical Treatment of Obstructive Lesions of the Esophagus. *Ann. Surg.*, 127:1067-1078, May, 1948.

30. Mautz, F. R.: Transpleural Resections of Esophagus and Stomach for Esophageal Obstruction. *Ohio State Med. Jour.*, 42:1243-1247, Dec., 1946.

31. Morton, D. R., and Brunschwig, A.: Peptic Ulcer of the Esophagus. Report of a Patient with 10-Year Follow-Up. *Gastroenterology*, 7:314-319, Sept., 1946.

32. Nagel, Gunther: Personal communication.

33. Olsen, A. M.: Esophagitis (Editorial). *Surg., Gynec. and Obst.*, 86: 372-374, Mar., 1948.

34. Olsen, A. M., and Harrington, S. W.: Esophageal Hiatal Hernias of the Short Esophagus Type: Etiologic and Therapeutic Considerations. *Jour. Thoracic Surg.*, 17:189-209, Apr., 1948.

35. Robins, S. A., and Jankelson, F. R.: Cardioesophageal Relaxation. *Jour. A. M. A.*, 87:1961-1964, Dec. 11, 1926.

36. Smithers, D. W.: Short Esophagus (Thoracic Stomach) and Its Association with Peptic Ulceration and Cancer. *Brit. Jour. Radiol.*, 18: 199-209, July, 1945.

37. Stephens, B.: The Problem of the Acquired Short Esophagus. Report of 18 Patients. *Calif. Med.*, 71:1-5, Dec., 1949.

38. Sweet, R. H.: The Surgical Treatment of Obstructive Lesions of the Esophagus. *Trans. Amer. Acad. Ophth. and Otolaryngol.*, pp. 118-125, Nov.-Dec., 1949.

39. Sweet, R. H.: Thoracic Surgery. Philadelphia: W. B. Saunders Co., 1950.

40. Van Aken, H.: Stenoserende Peptische Oesophagitis. *Nederlandsch Tijdschrift Voor Geneeskunde*, 94:944-947, Apr. 1, 1950.

41. VINSON, P. P.: Cicatricial (Benign) Stricture of Esophagus Following Vomiting and Intubation; Report of Three Cases. *W. Va. Med. Jour.*, 37:349-350, Aug., 1941.

42. VINSON, P. P., and BUTT, H. R.: Esophagitis. A Clinical Study. *Jour. A. M. A.*, 106:994-996, Mar. 21, 1936.

43. WANGENSTEEN, O. H.: Personal communication.

44. WANGENSTEEN, O. H., and LÆVEN, H. L.: Gastric Resection for Esophagitis and Stricture of Acid-Peptic Origin. *Surg., Gynec. and Obst.*, 88: 560-570, May, 1949.

45. WINKELSTEIN, A.: Peptic Esophagitis. A New Clinical Entity. *Jour. A. M. A.*, 104:906-909, Mar. 16, 1935.

46. WYNDHAM, N.: An Unusual Stricture of the Gullet Treated by Excision. *Med. Jour. Australia*, 2:724, Dec. 13, 1947.

PHYSICIANS' ART SHOW.

The American Physicians Art Association will have an art exhibit, as usual, during the A. M. A. Convention at Atlantic City, N. J., June 11 to 15, 1951, inclusive. Any physician in the United States, Canada and Hawaii desiring to participate in this show should communicate with the secretary for particulars.

J. Henry Helser & Co., Inc., Investment Managers with offices on the Pacific Coast, are the new sponsors of the American Physicians Art Association and will award 200 trophies besides a special Helser Trophy—a large decorative cup depicting Yankee ingenuity. This cup is to be awarded for art work done in any medium. Also, the large Popularity Trophy will be awarded to the owner of the art piece receiving the most popular votes during the A. M. A. Convention. Over 4,000 members of the American Physicians Art Association will receive shortly, entry blanks, shipping labels and rules about this fourteenth art exhibition.

The Annual Art Banquet will be held Tuesday evening, June 12, at the Marlborough-Blenheim Hotel, Atlantic City, N. J. Dr. F. H. Redewill, Secretary, 720 Market Street, San Francisco 2, Calif.

SURGICAL PROBLEMS INVOLVED IN THE REMOVAL OF GLOMUS-JUGULARE TUMORS.*

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Boston, Mass.,
and
CHARLES S. LANE, JR., M.D. (by invitation),
Fort Smith, Ark.

In 1941, Guild[1] described the glomus jugulare (approximately 0.5 x 0.25 mm. in size), lying in the adventitial layer of the dome of the jugular bulb or along the tympanic branch of the IXth nerve in its bony canal, or rarely as it lies in the sulcus of the promontory. This structure resembled the normal carotid body histologically and was presumed to have functions like it. Four years later, Rosenwasser[2] reported the first recognized tumor of Guild's structure, and called the neoplasm a "carotid body tumor" of the middle ear and mastoid. During the past five years a substantial amount of literature has appeared on this subject. Lattes and Waltner,[3] in May, 1949, were able to find a total of 18 cases of their own and others, while in August, 1949, Lundgren[4] reviewed 16, of which he himself had observed four. These two lists overlap and diverge in such manner that they total at least 27 cases. DeLisa[5] added one case in 1950.

Winship *et al.*[6] reported a cervical lymph node metastasis in one patient, while Lattes and Waltner[3] found one liver metastasis; otherwise all of the tumors have been either benign or only locally invasive.

The terminology is confusing, in that Lundgren[4] prefers "tympanic body tumor"; Rosenwasser,[2] "carotid body-like

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tumor"; Lattes and Waltner,[3] "nonchromaffin paraganglioma of the middle ear," while we believe that the term glomus-jugulare tumor is the most informative.

Although this is our preference, the term "nonchromaffin paraganglioma" has much to recommend it. It will be recalled embryologically that the central nervous system is derived from the ectodermal neural tube, and that from the ganglion crest of this tube are differentiated the spinal ganglia. Some of the cells of the ganglion crest migrate ventrally to form the sympathetic ganglia; however, others instead of becoming neurons develop into gland cells, as in the adrenal medulla, and are aptly called paraganglionic tissue. They stain brown with chrome salts, whence the term "chromaffin." The carotid body, about the size of a rice grain, has long been considered paraganglionic tissue, and has histological similarity to a sympathetic ganglion. The adrenal medulla and carotid body are two of many "pheochrome organs" scattered as paraganglia along the sympathetic nervous system. Watzka has been cited[3] as classifying the carotid body as nonchromaffin and nonepinephrine producing, yet paraganglionic; presumably the jugular body (glomus-jugulare) is in the same category. Guild[1] assumed the *function* of the glomus jugulare and carotid body to be similar. It will be recalled that the carotid body is an organ related to the reflex control of respiration and that its prime stimulus is anoxemia. It has nothing physiologically to do with carotid *sinus* reflexes regulating blood pressure.

In gross appearance, nothing is usually characteristic about glomus-jugulare tumors to distinguish them from ordinary inflammatory aural polyps, for which they are often mistaken. The fact that profuse hemorrhage may occur from the slightest trauma offers a clue to the correct diagnosis. In our Case 1 the Massachusetts General Hospital pathology laboratory rendered an interesting report regarding the gross pathology of the tumor examined there. "A rectangular segment of red-gray tissue measuring 1.7 x 1.0 x 0.8 cm. The material is spongy to palpation and is sectioned with fleshy resistance revealing red-gray moist cut surfaces from which

escape large amounts of fluid blood. No vascular channels are visible grossly." The histological picture explains the bleeding tendency, since the tumor consists of a meshwork of blood spaces, most of which have a single layer of endothelial cells separating them from cords and nests of tumor cells, which resemble those of sympathetic ganglia. Mitoses have not been reported.

The average case will, therefore, seem to have an aural polyp, bleeding unduly, with chronic suppurative otitis media and mastoiditis. An attempt to state definitive clinical characteristics has been made by Lattes and Waltner[3] as follows:

1. An extreme tendency to bleed.
2. Pulsating tinnitus synchronous with the heartbeat.
3. Progressive locally expansive type of growth producing areas of destruction in the temporal bone.
4. Possibility of metastases.
5. Long duration of symptoms with repeated polypectomies and recurrences.

It is the purpose of this paper to describe the practical operative problems encountered in dealing surgically with glomus-jugulare tumors and to report three cases that have been observed at the Massachusetts Eye and Ear Infirmary. One of these, having such a tumor present itself in both the ear and nasopharynx simultaneously, is of particular interest, in that no similar case has thus far been described in the literature.

SURGICAL PROBLEMS.

The surgical difficulties encountered in dealing with this locally invasive neoplasm are chiefly the risk of hemorrhage; recurrence; possible metastasis; danger of facial paralysis from local invasion or surgical trauma; and inability to employ conventional anatomical landmarks in the operation.

1. Hemorrhage is of prime importance. One reported case had a blood loss of about 1,000 cc.; a two-stage operation was required in three cases[3] because of formidable blood depletion at the first operation. It is, therefore, obvious that facilities for transfusion should be instantly available in the operating room, but much serious hemorrhage can be prevented by limiting direct trauma to the tumor to the shortest possible time; and by the *assumption* that it originates from the dome of the jugular bulb, from which the most violent bleeding is expected to occur. These objectives are accomplished by block dissection of the mass, and by deliberately leaving initially a small bit of the neoplasm in the floor of the hypotympanum. That is, bone is removed *to* the mass until it is widely exposed, avoiding direct trauma to the neoplasm. Rapid dissection is then done with removal of all gross tumor except in the floor of the hypotympanum. After obtaining a dry field, the residual tumor is teased away so far as possible without eliciting serious bleeding.

   This technique is somewhat analogous to that devised by Schall for the excision of highly vascular sinus tumors.

2. Recurrence is always possible, with or without invasion of neighborhood structures. It was not observed in the ear in our three cases, but all were followed for less than two years. Since the tumor grows slowly, we may have to face this problem later by means of further surgery, irradiation, or both.

3. Metastases have occurred in only two of 31 cases. It is our view that the danger of excision of the origin of the tumor in the juguar bulb offers a mortality risk infinitely greater than that of accepting the possibility of a metastasis. It will be recalled that the bulb underlies not only the floor of the middle ear but also the cochlea, and vestibule, and is in surgical contiguity posterolaterally with the facial nerve. Other relationships within the jugular foramen hardly require emphasis. In any

event, the glomus jugulare location may be quite far removed from the hypotympanum. To compress the lateral sinus and tie the internal jugular vein would not control blood flow from the inferior petrosal, or the arterial supply of the bulb from the ascending pharyngeal artery; therefore, we feel that the bulb must be spared.

4. Facial paralysis or weakness is of frequent occurrence in glomus-jugulare tumors. Our Case 3 had had a complete peripheral VIIth nerve palsy for eight years prior to admission to the Infirmary. It had followed a secondary radical mastoidectomy. It seems likely from the history that an unrecognized glomus tumor was present then.

   In listing all available cases in the literature and their own as 18, Lattes and Waltner[3] found 10 to have facial paralysis or weakness, with six preoperative and four postoperative. Gentleness in dissection of the region of the VIIth nerve is important while removing the main tumor mass and may seem difficult in the presence of profuse bleeding.

   The very thin horizontal portion of the Fallopian canal between the geniculate ganglion and crest of the horizontal semicircular canal must be given special care as a prophylactic measure. A paralysis from local invasion of the nerve may be a part of the disease, but an earnest effort should be made to avoid it from simple surgical trauma.

5. It is our belief that inability to employ the usual anatomical landmarks, specifically the horizontal semicircular canal, in the operation may account for an occasional postoperative facial paralysis. Almost as good orientation can be obtained by creation of the tympanic spines (spina tympanica major and minor) when the bridge is removed. These spines lie external and anterior to the Fallopian canal, and provide adequate orientation until the main tumor mass is excised, thereby exposing the horizontal canal.

The technique described below was utilized in Case 1:

The classical Lempert endaural incisions were used, and adequate exposure of the mastoid cortex was obtained. A perforating electric burr was used to create a triangular-shaped opening in the mastoid bone in McEwen's triangle, the apex of the opening being directed toward the mastoid antrum. An oval cutting burr or a round semipolishing burr was then used to deepen this defect until the antrum was found.

The antrum was filled with very vascular tumor tissue from which free bleeding issued. This was controlled by exerting pressure with small gauze strips. The location of the horizontal semicircular canal, which would have followed as the next step in a conventional radical mastoidectomy, was deferred. The reason for this was that any manipulation of the tumor tissue increased the bleeding. A search for conventional landmarks by removing this vascular tissue was not feasible.

The principle then used was removal of bone with an electric driven burr from around the tumor tissue without touching the neoplasm itself. In order to accomplish this, the posterior-superior canal wall was thinned down and lowered, and the bridge was removed. The anterior and posterior tympanic spines were then visible. Using these structures as landmarks, the removal of the posterior-superior canal wall was continued and the epitympanum was widened.

Removal of this bone did not entail traumatizing any of the tumor. The neoplasm was kept free of the burr by gentle pressure with a small elevator. Bleeding was thus kept at a minimum.

Though the horizontal semicircular canal had not been seen, the approximate course of the horizontal portion as well as the beginning of the descending portion of the facial nerve could be determined. With the position of the nerve in mind, removal of bone necessary to open the middle ear widely was continued.

Because the inner portion of the canal wall epithelium appeared to be involved with tumor, this portion of skin was sacrificed. This was accomplished by cutting in circular fashion across the skin of the canal outside the limit of extent of tumor tissue and then separating the skin from the bony canal wall from without inward to the tympanic sulcus. Then the remainder of the bone work at the margin of the middle ear space and external auditory meatus was completed with the electric burr.

There now presented a sizable block of neoplasm from the outer margins of which bone had been removed. Very gentle separation of this tumor tissue from the bone that lay deep to it was accomplished with a small Lempert periosteal elevator. When sufficiently freed, it was possible by rapid dissection to remove *en bloc* the mass which had filled the mastoid antrum, middle ear, and inner portion of the external auditory meatus. Free bleeding was encountered as the block was removed, but this was controlled by firm pressure with gauze packing. A small amount of neoplastic tissue was left in the floor of the middle ear cavity where the tumor appeared to arise from the jugular bulb. As much residual neoplasm as possible was teased away until violent but easily controlled hemorrhage, apparently from the jugular bulb, was encountered. It was not of practical value to remove more tissue in this region. Other than for this, it was felt that complete excision of the tumor was accomplished. A complete radical mastoid cavity had been created; it was then finished completely.

Nine days later, a tumor clinic consultation was requested regarding possible irradiation of the hypotympanic region. The specialist from the X-ray department there made the following answer: "The histology of glomus tumor arising in the ear is essentially that of glomus tumors elsewhere in the body. They are notoriously radioresistant, and there is no reason to presume that those in this location should be more responsive. The literature is quite wanting in any enthusiastic statement regarding the value of X-ray treatments, although a number have been recorded. Treatment is not

without hazard to the normal tissues in which the tumor lies, and I would not recommend this procedure. Even metastases have not been reported as having been favorably influenced by treating with X-ray."

On the seventeenth postoperative day, however, we decided to implant into the tympanum two radon seeds of one millicurie each. This was done under local anesthesia.

On the twenty-fifth day after operation the cavity was skin-grafted, and it healed uneventfully. Further details will be mentioned in the following case report.

CASE REPORTS.

Case 1: L. C., a 41-year-old white female, was referred to the Massachusetts Eye and Ear Infirmary on July 5, 1949, by a private physician, with a diagnosis of polyp of the left middle ear.

The history was as follows: Eighteen years previously she had developed a slight hearing loss in her left ear which had not noticeably increased until the months immediately following the birth of her fourth child on Dec. 11, 1945. At this time, she also noticed an increase in the quantity and frequency of a "buzzing" tinnitus which had occurred intermittently for several years.

In May, 1946, while she was removing cerumen from her left ear with a hairpin, the hairpin "appeared to get caught" in the ear canal, and when she removed it there was slight bleeding from the canal for a few minutes. During the following week she developed a purulent, foul-smelling otorrhea with a definite increase in the hearing loss in this ear.

In July, 1946, with the painless otorrhea still present, she first consulted a physician about the ear. Following his initial examination, the otologist informed her that she had a polyp in her ear, and subsequently she was admitted to a hospital, where the polyp was removed.

From that time until December, 1948, she had no further otorrhea, but the hearing loss in the ear slowly progressed. At no time was there any pain, nausea, vomiting, vertigo or headache. Occasionally she noticed a slight buzzing tinnitus localized in the left ear.

In December, 1948, while she was scratching the external auditory canal of her left ear with her fingertip, she felt a soft mass. This caused her to consult her physician, who discovered another polyp in the canal, and she was once more admitted to a hospital where the polyp was removed.

There was no aural discharge from that time until March, 1949. At this time, however, a slight otorrhea developed, which progressively increased in quantity and became foul; also, she began to experience otalgia which was stabbing and fleeting in nature. At no time did she have headache, vertigo, nausea or vomiting. Around the last of June, 1949, she again saw her physician, who told her that the polyp in her left ear had recurred. He then sent her to the Massachusetts Eye and Ear Infirmary.

The general physical examination was normal except for bilateral varicose veins of the legs.

Important local findings were limited to the left ear. Immediately inside the external auditory canal there presented the outer margin of a firm, grayish-white mass which completely occluded the canal. The canal was bathed with pus. The mass was slightly movable, and it appeared to be attached deep in the canal or in the middle ear. The auricle and mastoid were normal to inspection and palpation.

An air-conduction audiogram demonstrated normal hearing in the right ear and a 40 to 65 db loss in the left ear. The Weber test was lateralized to the left, and the Rinne test of the left ear was negative. Mastoid X-rays were reported: "Both mastoids are small. The left mastoid is increased in density."

Thirty cc. of iced alcohol syringed into the left external auditory meatus produced no nystagmus or vertigo. The fundi were normal; the neurological examination was not remarkable. X-rays of the base of the skull failed to demonstrate any bony erosion. X-rays of the hands, feet, pelvis and hips were negative for possible metastasis.

On July 12, 1949, a large portion of the mass presenting in the left external auditory meatus was removed with an aural snare. Profuse bleeding from the canal followed removal of this tissue, and required firm packing with gauze strips for adequate control. There was no bleeding when the packs were removed the following day. The pathological report of the biopsy was "glomus-jugulare tumor."

A left endaural radical mastoidectomy with removal of the tumor was then performed as previously described.

The patient's postoperative course was entirely uneventful. At the end of a month, she was ready for discharge. The cavity at this time had a very satisfactory configuration; the graft had taken well; the ear was dry. There had been no recurrence after a 13-month follow-up, and the cavity had remained dry.

Case 2: L. M., a 57-year-old white female, was first seen at the Massachusetts Eye and Ear Infirmary in September, 1944, complaining of discharge from the right ear for nine years. There had been an occasional attack of dizziness, but no true rotary vertigo, and there had been no headache, nausea or vomiting. She had noticed gradual progressive hearing loss in her right ear. She had also experienced several bouts of hemorrhage from the right ear which she had been able to control at home with cotton packs.

The general physical and neurological examinations were normal.

The local examination revealed the external auditory meatus of the right ear to be filled with a granular mass which bled when manipulated. Otherwise the local findings were not remarkable.

X-rays of the mastoids were reported as follows: "Both mastoids are small. The right mastoid is sclerosed; the left mastoid is pneumatized and clear."

On Sept. 19, 1944, the mass in the right external auditory meatus was partially removed by means of an aural snare. This procedure was accompanied by rather profuse hemorrhage from the canal, which was controlled by adrenalin packs. The hemorrhage recurred the following day, but the packing was removed the second postoperative day, without further bleeding. Biopsy report was "hemangioma."

The patient was discharged to the Out-Patient Department where she was subsequently followed. During the following month, the aural discharge ceased, and the remaining mass of tumor tissue deep in the external auditory meatus became covered with epithelium.

Caloric tests performed at this time demonstrated active labyrinthine reactions on both sides. The fistula test was negative.

The patient was not seen again until January, 1949. She then reported to the out-patient department complaining that her right ear had started discharging again several weeks after her last visit and had continued to discharge since that time. There had also been several bouts of slight bleeding from this ear, and three days before, she had experienced a rather severe hemorrhage which she had controlled with cotton packs.

She was readmitted to the Infirmary on Feb. 2, 1949.

General physical examination again was essentially negative. On local examination, the right external auditory meatus was found filled with a freely movable, smooth, grayish-white polyp-like mass. The fistula test was negative. With the left ear masked, she had no hearing in the right ear.

Mastoid X-rays were reported: "The left mastoid is normal. The right mastoid has a few cells showing increased density. There is no bone destruction."

A biopsy was taken by removing a portion of the mass in the left external auditory meatus. Profuse hemorrhage resulted, which necessitated tight packing of the canal with gauze.

The pathological report of this biopsy was "glomus-jugulare tumor."

On March 8, 1949, a postaural radical mastoidectomy with removal of the tumor tissue was performed.

On March 16, 1949, the packing was removed with only slight bleeding, and the cavity appeared to be in good condition. On March 27, 1949, she was discharged and was subsequently followed in the Out-Patient Department.

Early in April, 1949, it was noted that the cavity appeared to be filling in with granulations, but the bowl was almost dry. Subsequently, the bowl became smaller and entirely lined with smooth epithelium. It was completely dry, and remained so when she was last seen, 22 months following her operation. There had been no recurrence observed.

Case 3: G. D., a 37-year-old white female, was first admitted to the Massachusetts Eye and Ear Infirmary on April 29, 1949, with a diagnosis of a mass in the nasopharynx.

Her previous history follows: In 1933, the patient developed a painless purulent discharge from her left ear. During the next several years, she had a progressive hearing loss in this ear, experienced recurrent exacerbations and remissions of the otorrhea, but she never had any pain, vertigo, nausea or vomiting. Treatment consisted of local cleansing of the external auditory meatus, the use of ear drops, and on several occasions "aural polypectomies" were performed.

On Aug. 14, 1940, the patient had a left postaural radical mastoidectomy. Following this operation, she continued to have left otorrhea. On July 1, 1941, there was a revision of the radical mastoidectomy following which the patient had a complete peripheral paralysis of the left facial nerve. The cavity became dry, but the facial paralysis persisted.

In 1946, she began to have bouts of mild, easily controlled epistaxis. The nasal obstruction was such that she was unable to get any air through the left side of her nose, and the right side was almost completely blocked also.

When she entered the hospital, her general physical examination was normal.

On local examination there was a complete peripheral left facial nerve paralysis. The plastic opening into the left radical mastoid cavity was markedly stenotic, and the cavity was moist. It contained a small amount of polyp-like tissue. There was a well-healed left postaural scar. Anterior rhinoscopy was normal, but examination of the nasopharynx revealed a firm, round, smooth, grayish-white mass completely filling the left half of this space. The mass appeared to have its origin from the superior part of the fossa of Rosenmüller.

X-rays of the nasopharynx and temporal bones were reported: "There is a mass in the left upper part of the nasopharynx which is very suggestive of malignancy. The right mastoid is normal. The left mastoid is postoperative. There is destruction of the lower part of the petrous bone —below the cochlea—which appears to be involved."

On April 30, 1949, a biopsy was taken from the lower limit of the mass in the nasopharynx, which included approximately one-fifth of the visible substance of the tumor. A profuse postnasal hemorrhage resulted, which required packing of the nasopharynx and left nasal cavity.

The pathological report on this tissue was "angioma, suggestive of glomus tumor."

Two days after the biopsy was taken, all packing was removed without further bleeding.

On May 3, 1949, the patient was discharged from the Infirmary to the Out-Patient tumor clinic. Here, during the next few weeks, a rapid resolution in the size of the mass was observed, so that by the end of May, 1949, the entire nasopharynx appeared free of tumor, and only a denuded area high in the fossa of Rosenmüller on the left side could be seen. She had experienced no further epistaxis, and she was breathing easily through both sides of her nose.

On June 20, 1949, the patient was readmitted to the Infirmary for investigation of the polypoid-like mass previously noted in the left mastoid cavity. A biopsy of this tissue on June 22 was reported as "glomus-jugulare tumor."

On June 27, 1949, an endaural revision of the left mastoid cavity with removal of the tumor was performed.

Pathological study of the specimen obtained from the middle ear region confirmed the original diagnosis of glomus-jugulare tumor.

The subsequent course of the patient is interesting. She was discharged two weeks after her operation. The mastoid cavity required about three months to become covered with epithelium; at first it seemed to fill with granulations, but then shrank after an epithelial covering grew in. It appeared

to be shallow and stenotic, but various observers found no evidence of tumor recurrence in the ear up to and including the patient's last visit 18 months postoperative.

The nasopharynx remained free of any sign of neoplasm for nine months. Then a small purplish-red area of discoloration appeared at the orifice of the left Eustachian tube. This increased slightly in size during the next 11 months of observation, but the patient remained free of symptoms and no treatment was deemed necessary. She had gained 10 pounds since her operation.

SUMMARY.

Some of the practical surgical problems encountered in dealing with glomus-jugulare tumors are considered and three additional cases with this diagnosis are discussed.

One glomus-jugulare tumor presenting itself simultaneously in both the ear and nasopharynx has never been previously reported in the literature. In this case there was destruction of the petrous bone below the cochlea demonstrable on X-ray examination; an angiomatous neoplasm "suggestive of glomus-jugulare tumor" attached to the vault of the fossa of Rosenmüller on the side of a proven glomus-jugulare tumor in the mastoid. Anatomically, the extension of the lateral superior aspect of the nasopharyngeal wall as far as the anterior margin of the carotid foramen signifies with the above evidence that the "two" tumors were one and the same.

BIBLIOGRAPHY.

1. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus-Jugularis in Man. Abstracted, *Anat. Rec.*, Supp. 2, 79:28, 1941.

2. Rosenwasser, H.: Carotid Body Tumor in the Middle Ear and Mastoid. *Arch. Otolaryngol.*, 41:64, Jan., 1945.

3. Lattes, R., and Waltner, J. G.: Nonchromaffin Paraganglioma of the Middle Ear. *Cancer*, 2:447, May, 1949.

4. Lundgren, N.: Tympanic Body Tumors in the Middle Ear. *Acta Otolaryngol.*, 37:367, 1949.

5. DeLisa, D. A.: Tumor of the Glomus Jugularis. *Arch. Otolaryngol.*, 51:925, June, 1950.

6. Winship, T.; Klopp, C. T., and Jenkins, W. H.: Glomus Jugularis Tumors. *Cancer*, 1:441, Sept., 1948.

RHINOGENIC BRAIN ABSCESS.*

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Rhinogenic brain abscesses have their origin about the nose, nasal cavities and paranasal sinuses, but brain abscesses originate from infected middle ears, mastoid, accessory nasal sinuses, the calvarium; from penetrating wounds or from metatases. As we are interested only in those abscesses which have their origin from a rhinogenic source, the discussion will be based on that premise.

ETIOLOGY.

The etiological factors are *1.* acute upper respiratory infections, especially those which produce acute purulent sinusitis; *2.* chronic sinusitis with an acute exacerbation, which becomes purulent with lack of drainage; *3.* extraction of teeth, which may produce a purulent maxillary sinusitis; *4.* fractures of the skull involving the sinuses; *5.* puncture wounds involving the nose, nasal cavities and sinuses; *6.* surgery in acute or green sinusitis producing osteomyelitis of the skull; and *7.* furunculosis involving the anterior nares or angle of the nose.

ROUTES OF INFECTION TO INVASION OF THE CRANIAL CONTENTS.

Invasion may be by continuity through bony defects consequent to osteomyelitis; local erosion of bone or fracture; through congenital defects in adjacent bony walls; by extension through preformed pathways; by way of blood vessels, especially veins; by way of perineural sheaths. The lymphatics may play a part in the routes of infection, according to some authors. Infection, which has reached the pia mater

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from an adjacent focus, usually gains entrance to the brain by way of an entering artery, an emerging vein, or the perivascular spaces. Because of its more abundant blood supply the cortex does not yield to infection which extends into the white matter. The first stage of an abscess may be an infarct with arterial occlusion, hemorrhagic softening, or venous thrombosis. The type of this primary lesion probably disposes to the type of abscess which is to develop. As infection spreads in the decadent tissues, the clinical symptoms are those of encephalitis.

BACTERIOLOGY.

The causative organisms most frequently found in brain abscesses are as follows: staphylococcus aureus hemolyticus, streptococcus hemolyticus, pneumococcus, staphylococcus aureus, staphylococcus albus hemolyticus, streptococcus sub acidus, Gram negative bacilli, and streptococcus sub acidus hemolyticus. Anaerobic organisms do not favor the formation of a capsule in a brain abscess while aerobic organisms favor the formation of a capsule.

PATHOLOGICAL FINDINGS.

Histological examination of brain abscesses reveals four zones of reaction; the inner zone of purulent material, the fibrous zone, the reactive zone of cellular structure and the encephalic edematous zone. The inner zone is a necrotic zone composed of polymorphonuclear leucocytes, tissue debris and, in long standing abscesses, deposits of calcium. This zone is surrounded by a reactive zone which is composed of loose connective tissue in which lie fibrin vessels and numerous cells; namely, polymorphonuclear leucocytes and lymphocytes. The reactive zone is surrounded by a dense layer of fibrous tissue designated as a fibrous zone, and this is surrounded by a zone of polymorphonuclear cells in early stages with a few lymphocytes and plasma cells, and later with edema in the encephalic zone. Underlying the abscess is a large or small area of meningitis depending upon the size of the focus.

Symptoms:

General

Headache
Convulsions
Mental changes
Bradycardia
Vomiting
Dizziness

Local

Palsy of contralateral VIIth nerve
Hemianoptic fields of vision
Aphasia
Papilledema
Hemiparesis
Orbital edema
Ocular motor palsy
Abducens palsy

ROENTGENOLOGICAL FINDINGS.

Roentgenological findings reveal changes in the osseous structure of the sinuses and the skull. Spinal fluid changes are not too great.

Diagnosis

History of case
Physical examination
General and local
General symptoms
Local symptoms
Laboratory findings
Roentgenological
Spinal fluid
Bacteriological

Treatment

Depends on what is found at examination
Expectant — medical
Surgical

Prognosis depends upon the status of the capsule formation. The increased intracranial pressure should be relieved by a decompressive osteoplastic flap with the excision of the abscess after a period of weeks when the capsule has become sufficiently strong to permit its complete removal without rupture. At present this method is thought to bring about good results in the way of a cure. Of course, if the abscess has been located, and drainage is established, the same results may be obtained as by decompression.

Heavy encapsulated abscesses have been carried for months without having been suspected of being brain abscesses, but it was thought at the time of operation that the surgeons were dealing with some other type of brain tumor.

1. How can one determine the status of the capsule?

   a. By the length of involvement.

   b. By diminution in signs and symptoms. Falling temperature, decreasing leucocytosis, stabilization of papilledema and an improvement of the general condition of the patient.

TREATMENT OF THE INFECTIOUS SOURCE OF THE ABSCESS.

Many cases improve under hypertonic salt solution for a period of time, but this cannot be continued for too long a period for fear of producing toxic dehydration. These cases later develop the same symptoms which they had at first, but during this period of improvement enough time has elapsed for the formation of a capsule.

In many cases of brain abscess the original source of infection has been eradicated or drained before coming to the neurologic surgeon. This was thought to be the better procedure, but recently some neurologic surgeons have thought it better to eradicate the abscessed cavity first, and the source of infection later. Personally, I would prefer the former method in most cases.

From a surgical standpoint if there is a definite capsule formation, the abscessed cavity should be removed as though it were a tumor; in fact, that is what it really becomes as far as symptomatology is concerned.

I am presenting a case of frontal lobe abscess which we first saw at Indiana University Hospitals in April, 1950.

B. M. R., a 30-year-old man, was admitted to the Robert Long Hospital, April 10, 1950, and was discharged May 25, 1950. History revealed that the patient became acutely ill about three weeks prior to admission following "postflu" when he began to have a swelling of the left eye, with pain above the left eye, in and around the orbit. This was accompanied by chills and fever. One week prior to admission the patient was placed on penicillin and developed a right hemiplegia.

Examination revealed a doughy supraorbital edema above the left eye with marked swelling and redness of the eyelids and orbit, so that the lids were entirely closed. There was paresis of the left oculomotor muscle. The pupils were small, equal with some irregularity on the left; no papilledema. The neck was stiff. He could not open his mouth well, at least not sufficiently to protrude his tongue. There was a right facial palsy with paresis of the right arm and right leg. Abdominal reflexes were absent. Examination of the nose revealed it to be full of pus. Reflexes on the right were hyperactive; Babinski was negative; there was no clonus. The patient was aphasic.

Lumbar puncture was done and opening pressure was 170, closing pressure 120; the fluid was clear and sparkling. Urinalysis was normal. Complete blood count showed hemoglobin 13, W.B.C. 10,350, bands 5, polys. 60, lymphocytes 27, monocytes 5, eosinophiles 2, basophiles 1. Spinal fluid showed red cells 30, white cells 11, globulin (Pandy) 2 plus, sugar 39, protein 124, serology negative, gold curve 3445555554. Spinal fluid culture revealed no growth.

X-ray studies showed normal chest. Films of the skull showed normal sella turcica, normal calcified pineal gland, no evidence of osteitis in the calvarium, petrous ridges and mastoid areas normal. The dorsal sella was normal. Films of the paranasal sinuses showed definite opacity of the anterior and posterior ethmoidal cells on the left and of the entire cavity of the left maxillary antrum with less translucency than that of the right. No bone destruction was detected. There was marked deviation of the cartilaginous portion of the nasal septum to the left. Loss of contrast over the frontal area was attributed to swelling of the scalp and forehead overlying this area.

Lateral films of the mastoids were normal.

On the day of admission the patient was placed under the care of Dr. R. J. McQuiston and the chief resident. He was taken to surgery and with incision through the left eyebrow in midportion of the orbit, the periosteum was elevated over the frontal sinus and a large subperiosteal abscess was encountered which exuded a large amount of pus. The orbital plate was found to be broken through and was removed. The cranial plate of the frontal sinus was examined and appeared in good condition. The opening was made through it 1.5 cm. from the orbital margin and good dura was seen. At this time a small bead of pus was seen to form

and re-form at the upper outer margin of the frontal sinus. This opening was enlarged and was followed by a larger gush of pus estimated to be about an ounce. The orbital plate was found, removed and a brain abscess stalk was seen. A Pezzer catheter was passed over a stylet into the abscess cavity and the wound was sutured with silk. A left antral window was done using a rasp and antrum punches. Culture of the pus revealed strep. viridans.

Postoperatively the patient remained mentally clouded for several days, but gradually improved with improvement in his neurological picture with return of some function in his right-sided paresis. On April 18, 1950, a lipiodol injection through the catheter drain revealed an abscess of the frontal lobe area measuring approximately 2 by 2.5 by 3 cm. There was gradual improvement until April 27, 1950, 17 days after admission. At this time the aphasia became worse, the hemiplegia seemed to remain static. Visual fields at this time seemed normal. Neurological examination showed involvement of the left third with some resolution of the right seventh. Deep reflexes were hypoactive. No clonus was present; Babinski and Hoffman were negative. No sensory changes could be found.

Progress X-ray films indicated the cavity to be 4 by 5 cm., indicating an increase in size, and there was apparent sclerosis of the left frontal sinus in the frontal plate area which probably represented reactive bone to osteomyelitic involvement of the frontal plate.

On May 1, 1950, the patient was referred to the neurological staff for surgery which was carried out by Dr. Robert Heimberger and staff. A caronal incision over the frontal area was made, the periosteum was turned downward forward. A left bone flap was raised using five trephine holes and the dura was exposed. The dura was found to be densely adherent to the arachnoid over the area of the frontal lobe. Once the dura was incised and lifted, the brain was explored with a ventricular needle and the abscess was found in the frontal lobe. An incision was made in the cortex; this was carried downward and the lining of the cavity exposed. The entire lining of the abscess was then removed. The wound was thoroughly irrigated, the dura was sewn in place, the bone flap was replaced and kept in position by suturing the periosteum.

Pathological report was as follows: Microsection: *a.* The lining of the brain abscess shows the features to be expected, *i.e.*, a central area of liquefaction necrosis with little or no architecture remaining. This is lined by a zone of fibroblastic activity and glial proliferation in which small numbers of polymorphonuclear leucocytes can be identified. There is also an increased vascularity of the wall with many thin-walled vascular channels in the thicker tissues at this site. In the central area of liquefaction necrosis numerous small basophilic bodies are encountered which are approximately the size of bacteria. Most of these appear coccoid. They may, however, represent tissue debris, but since both streptococcus viridans and staphylococcic epidermidis were isolated, these could well present bacterial colonies. *b.* The specimen from the cortex shows a congestion and edema. The meninges show the presence of a subacute or chronic meningitis with fibrous proliferation and increased vascularity as well as infiltrating cells, both lymphocytes and polymorphonuclear leucocytes.

Interpretation: This brain abscess is a typical picture and is associated with a meningitis as well.

Pathological and Bacteriological Diagnosis: Brain abscess (due to strep. viridans and staph. epidermidis, bacteriological cultures). Meningitis, subacute (presumably some etiological agent).

In the early morning following surgery, the patient suddenly went into a shock-like state, was cold, clammy with a pale color and drowsy. He was given a blood transfusion and improved.

By May 5, 1950, four days postoperatively, the patient was still aphasic but the wound was healing well. There was still some drainage from the sinus tract over the eye. The patient was rational, temperature was normal, he was eating and appeared much improved. By the time of discharge, May 25, 1950, there had been further improvement in his hemiparesis, the patient had begun to talk, the sinus tract had ceased draining and was healed and the patient seemed mentally competent.

On June 9, 1950, the patient was seen in the out-patient clinic. Two days prior to his visit he had suffered a Jacksonian type right-sided convulsion, following which he slept most of the rest of the day. Examination revealed right facial, hypoglossal paralysis which had almost disappeared. The patient's muscle power in the extremities was almost equal, there were no sensory changes, reflexes were slightly decreased on the right compared to the left. The patient was started on dilantin sodium and phenobarbital.

The patient was followed periodically in the out-patient clinic. The seizures occurred about one per month until in October when they increased in their frequency so that by November the seizures were occurring at about one per week, and the patient was suffering weakness and mental confusion accompanying these seizures.

The patient was readmitted Nov. 7, 1950, with discharge Nov. 18, 1950. Electroencephalogram showed S_2 with greater slowing at the left frontal temporal.

Physical examination on admission revealed a man with some difficulty of speech and inability to express himself. He seemed somewhat confused as to time and place. Neurological examination was negative except for some residual right, mild hemiparesis. Lumbar puncture showed clear spinal fluid and normal dynamics and normal chemistry.

Films of the paranasal sinuses showed the previous surgical defect, but no new developments. Pneumoencephalogram showed dilatation of the left lateral ventricle, especially of the anterior horn, suggesting herniation into the area previously occupied by the brain abscess. The ventricular system otherwise was normal.

While in the hospital on regular foods and medication, the patient had no seizures. Just prior to the last admission the patient had been quite a problem at home with temper tantrums and being easily upset.

The patient was last seen in the out-patient clinic Dec. 1, 1950. He had had only one convulsion since discharge from the hospital, and this had been accompanied by a marked increase in his hemiparesis which cleared up within a matter of hours thereafter. He had been somewhat less irritable but still became upset with the children; was able to help a little with housework. He was still aphasic but seemed somewhat improved and appeared to be primarily expressive. The left pupil was slightly larger than the right. Reflexes could be obtained more easily on the right than left but there was not much disparity.

The patient was continued on dilantin sodium and phenobarbital, and it was felt that he could probably be ready to attempt part-time work soon.

SUMMARY.

1. Rhinogenic brain abscesses are rare.
2. I am quite sure antibiotics will further reduce their frequency.
3. The symptomatology is practically the same as other brain abscesses, especially if they are cerebral in type.
4. Encapsulation is necessary for recovery.
5. When well encapsulated, the abscess should be removed.
6. Aerobic organisms favor encapsulation of the abscess, and anaerobic organisms do not.
7. There is a question as to whether the source of infection should be eradicated first, or whether the abscess should be removed first.
8. Finally, a case is presented which presents all the symptoms which go with a brain abscess.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 1951-1952 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

FACIAL FRACTURES. CASE PRESENTATION.*

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Los Angeles, Calif.

The mandible is a modified long bone,[1] sheathed in a reinforced mucoperiosteal coat. Fractures of this bone occur approximately four tmes oftener than those of the maxilla.[2] In contradistinction to other long bones, mandibular fractures tend to shatter. (This may, in part, be due to the snug mucoperiosteum exerting cortical pressure against the solidly contacted dental arch, thereby allowing for a minimum of cushioning of an exciting force.)

Two muscle groups and tendons control the mandible's function. The larger posterior group includes the temporal, masseter, the internal pterygoid and two-bellied external pterygoid muscles. The small anterior group lies mostly in the mouth floor and incorporates the mylohyoid, the geniohyoid, geniohyoglossus and the two-bellied diagastric muscles. Both muscle groups pull the mandible toward the midline, but the posterior group pulls upward while the anterior group pulls downward.

In this consideration of facial fractures, without loss of substance, local factors influencing displacement of fracture fragments (of the mandible and maxilla) are: the number, position and character of the teeth; the nature of the causative force; the bevel of the fracture lines, and the extent of soft tissue damage.

Because teeth serve as the principal structure for immobilization, mandibular fractures may be classified as:

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1. Teeth upon both sides of the fracture line. *2.* Teeth upon one side of the fracture line. *3.* Teeth absent upon both sides of the fracture line.[2]

Fixation of reduced mandibular fragments is accomplished by either the intraoral or the extraoral route, or a combination of both.

A. INTRAORALLY:

1. Wiring of the teeth is most universally used. It may be:

a. Horizontal—that is, confined to one arch.

b. Vertical or intermaxillary—that is, immobilizing the two arches in approximation. (Occlusion here being the ideal approximation.)

2. Utilization of an orthodontic band—arch appliance, or

3. Capping the teeth and soldering the caps to form a splint, and

4. Lastly, utilization of an acrylic splint.

B. EXTRAORALLY:

1. Roger Anderson's pin fixation.

2. Kirschner wire.

3. Head band appliance.

Simple fractures seen early should require only wiring. Compound fractures are relatively frequent. Extensive soft tissue damage may require constructing splints that prevent secondary contractures. This serves both esthetic and physiological purposes.

In fractures of the angle, wherein the ramus is depressed medially, a convenient device to hold the ramus laterally is a piece of shoehorn bent at an off right angle. This is fixed buccally to the intermaxillary wires, after the posterior flange has been adapted to the lingual surface of the ramus. This obviates external pin fixation. Many conservative operators use external pin fixation only when other measures are ineffective.

In children, interosseus wiring is advisable. Fine wire minimizes damage to the permanent tooth buds that occupy a large portion of the tooth-bearing segments.

Fractures of the maxillae are usually associated with those of other facial bones. Displacement, if present, occurs here, at the time of injury, because only the pterygoid muscles exert a substantial pull upon these bones.

Because of weak points that invite stress, fractures of this bone tend to follow definite patterns. Lefort recognized this in classifying three fracture types.[3]

Type 1: Bilateral maxillary fractures are frequently transverse. They pass through the tooth-bearing segment, including the palate, base of the nose and the medial and lateral orbital walls. Downward displacement may press upon the dorsum of the tongue. Upward displacement may impact into the antrum and fix the fragments between the malar bones.

Type 2: In the middle third fractures the line usually passes through the lateral antral walls, infraorbital canals, orbital floor, medial orbital walls and the nasal-frontal suture. This fragment, also, may displace upon the tongue or impact between the malar bones.

Type 3: The bones of the middle part of the face may here become detached from the skull, carrying along the maxilla's pyramidal process and its articulating zygoma.

Combinations of transverse and vertical fractures may be present. Treatment, to be adequate here, must restore facial contour as well as occlusion. Immobilization may be secured with intermaxillary wiring, retention splints, or plaster skull caps, the latter serving for anchoring of forked splints such as the Kingsley splint.[4] Impacted fractures may be mobilized with forceps. Cutting of adhesions facilitates moving older stabilized fragments.

The zygoma articulates with the frontal, sphenoid, temporal and maxillary bones. It forms the outer one-half of the orbital floor, enters into formation of the zygomatic and temporal

fossa and gives attachment to the temporal and masseter muscles. This triangular shaped bone with its three processes helps buttress the maxilla on its lateral aspect.

Simple zygomatic fractures initially may be obscured by swelling, and deformity noted later, after the edema has subsided. Fragments when pushed medially and downward may rotate, thereby separating the frontal and sphenoid articulations. In the orbital cavity this may carry both the suspensory and palpebral ligaments inferiorly, stretch Tenon's capsule and result in diplopia in the first instance, and ptosis of the lateral canthus in the second instance.

Depressed fractures may ride upon the mandible's coronoid process, thereby preventing closure of the mouth.

Comminuted fractures may splinter the orbitoantral party wall, the fragments thus impinging upon either or both of these cavities. A hematoma may also complicate these fractures.

Treatment is facilitated by a local or light general anesthesia such as sodium pentothal. A combination of both may be used. Anteriorly placed depressed fractures may be elevated without incision by using simple traction with a hook or tenaculum; however, a more direct approach through a buccal vestibule incision facilitates easy reduction and permits great leverage to be exerted.

When the temporal bone's zygomatic process is also fractured along with the zygoma, the temporal muscle route affords the most direct approach for reduction. Cleaning out a hematoma or removing bony splinters or replacing and packing fractured walls may be most advantageously done through a radical maxillary antrum operation. Here counter drainage through an intranasal antrum window is advisable.

Open reduction with wiring, or introduction of a bone graft may be necessary. Especially is the former advantageous for maintaining the frontozygomatic contour with wiring.[5]

In the nose, the adult's nasal bones fuse in the midline. Hence, in contradistinction to fractures in children, fractures

here are often bilateral. The Manhattan Eye and Ear Hospital in a series of 190 nasal fractures reports that 80 per cent occurred at the junction of the thin bony tip with the thickened body.[2] This point may be identical to the place where the upper border of the underlying upper lateral cartilage terminates cephalically.

Frontal forces sufficient to comminute the nasal bones may fracture the underlying septum. Lateral forces may cause one nasal bone to impact under the maxilla's frontal process, or the whole bony arch may at another time be swung free of all its articulations.

In the cartilaginous portion of the nose, the upper lateral cartilages' alae fuse with the septum. The lower lateral cartilages, however, are free and over-ride the upper lateral cartilages; hence injury, dislocation or fracture of the bony nose also involves the cartilaginous framework to a more or less degree.

Simple fractures seen early are easily repositioned under either a local or light general anesthesia. An elevator, swathed with cotton, moistened with saline and dipped in vaseline usually suffices for elevation. Impactions may require using an Ash or Walsham's forceps. The above procedures should suffice for contour restoration; however, attempts to realign the septum are more difficult. Especially is this true at the osteocartilaginous junctions, and particularly on the palatomaxillary floor. To satisfy best physiological results, submucosal replacement of the displaced osseocartilaginous tissues is often indicated. Stabilization of the displaced tissues is usually accomplished with vaseline strips, firm enough to maintain fragment position but loose enough to facilitate adequate circulation. Externally a cushioned stent dressing, anchored by adhesive, will maintain good immobilization for 24 to 48 hours. Comminuted fractures when not satisfactorily treated by the foregoing, may be suspended from a frontal head piece with a Kazanjian splint.

Complications of facial fractures are the exception. Rarely a basal skull fracture may be present. This may be noted by

bleeding from an ear canal, but it is to be remembered that trauma from a condyle may also produce bleeding here. Cerebrospinal rhonorrhea may be proof of intracranial injury.

Primary arterial hemorrhage in the floor of the mouth is often troublesome until the offending artery, usually the lingual, is ligated. A hematoma may complicate a nasal fracture and is best evacuated early. Secondary orbital and lacrimal adhesions may occur, and require subsequent plastic procedures.

Malocclusion may require correction later. Infection, particularly with anaerobic organisms, used to be a dreaded complication, but chemotherapy and antibiotics have largely obviated it, with its sequela, osteomyelitis.

Delayed or nonunion, except in the aged, is usually of mechanical origin.

Although traumatic facial fractures are increasing, contemporary medical care is effecting a much better anatomical, physiological and cosmetic result. Too much credit for these good results cannot be given to our system of free enterprise in American medicine.

CASE PRESENTATION.

V. S., a 45-year-old white female, was perfectly well until Oct. 26, 1950, when, between the hours of 12:00 and 1:00 P.M., while riding in an automobile she sustained head injuries rendering her unconscious. Emergency room examination revealed multiple contused facial wounds, a dislocated nose, a tongue that was slit back from the dorsum's tip for 5 cm. The nose was dislocated to the left, and a compound fracture was present in the body of the left mandible. Blood pressure was 160/85, pulse 88, fair quality, respirations 12 per minute and regular. Treatment for shock was instituted and a Barton bandage applied for mandibular immobilization. Subsequent history and physical examination were noncontributory.

Qualitative urinalysis was negative. The blood examination showed hemoglobin 11.7 gm. per 100 cc., red blood cells 4,010,000, leucocytes 7,250. The differential blood count showed polymorphonuclears 59, lymphocytes 40, monocytes 1.

Roentgenograms of the skull and chest were negative except for a fracture through the angle of the mandible on the left side. The anterior fragment was medially displaced and the posterior fragment over-rode it about 7 cm. After the patient gained consciousness and was out of shock, the soft tissues were cleansed, approximated and sutured, both intra- and extraorally.

Ten days later, after the orbital and liqual edema had subsided, because of difficulty in breathing through her nose, a rhinoplasty was done and the displaced upper and lower cartilaginous obstruction rectified.

Nine days later an unsuccessful attempt at a closed reduction of the mandible was attempted under local and light general anesthesia. An open reduction was then done through a left suprahyoid approach. The adhesions were severed, the fragments approximated and fixed with 22 gauge silver wire.

Because of an endentulous upper jaw an acrylic upper denture, previously made, was placed *in situ*. This acrylic denture had labial and buccal hooks to which the bands from the remaining lower teeth were attached, thereby completing intermaxillary immobilization. A Kingsley splint, which also had been previously affixed to the acrylic upper denture, permitted stabilization and fixation of the upper denture to a plaster headband, thereby immobilizing both jaws. This appliance was removed after six weeks, Roentgenograms showing a good callus formation.

This case was interesting for the following reasons: Initially the oral and glossal edema in the presence of nasal obstruction caused dyspnea, and prevented immediate immobilization of the fractured mandible. It was first necessary to permit the buccal edema to subside, then to restore the normal nasal airway, before reducing the fractured mandible. Delay in reducing the mandibular fracture permitted a fibrous union to form which made it necessary to use open reduction. Had the mandible been reduced earlier in the presence of nasal obstruction, it might have been necessary to resort to a tracheotomy.

BIBLIOGRAPHY.

1. Wilensky, A. O.: Osteomyelitis, Its Pathogenesis, Symptomatology and Treatment. New York, N. Y.: The MacMillan Co., 1934.

2. Kazanjian, V. H., and Converse, J. M.: The Surgical Treatment of Facial Injuries. Baltimore, Md.: The Williams & Wilkins Co., 1949.

3. Le Fort, R.: Etude Experimentale sur les Fractures de la Machoric Superieure. *Rev. d. Chir.*, 23:208, 360, 479, 1901. Quoted by Kazanjian in The Surgical Treatment of Facial Injuries. Baltimore, Md.: The Williams & Wilkins Co., 1949. (After Kazanjian, V. H., and Converse, J. M.)

4. Fry, W. Kelsey, Shepard, P. Rae; McLeod, Alan G., and Parfitt, Gilbert J.: The Dental Treatment of Maxillofacial Injuries. Philadelphia, Pa.: J. B. Lippincott Co.

5. Converse, J. M.: *Ann. Otol., Rhinol. and Laryngol.*, 52:637-663, 1943.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

May 1, 1951.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.
Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Distributor: Sears-Roebuck Co., 925 S. Homan Ave., Chicago, 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 400; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
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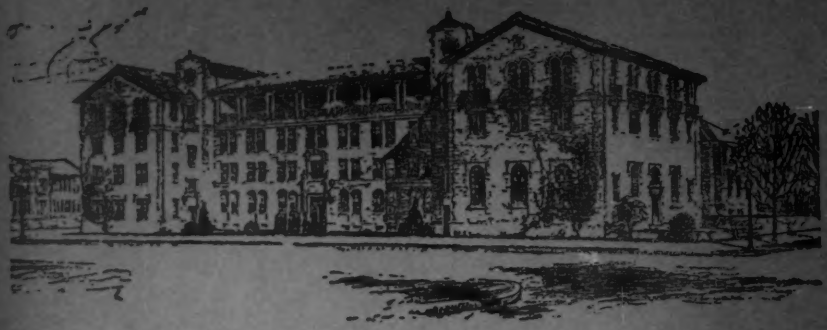

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